A DISSERTATION ON

THE BACTERIOLOGY OF PLEURAL SPACE INFECTION AND CLINICAL, LABORATORY AND PHYSICAL DETERMINANTS OF OUTCOME OF INFECTION

Submitted to

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

CHENNAI

In partial fulfillment of the regulations

for the award of the degree of

M.D. BRANCH - XVII

PULMONARY MEDICINE

GOVT. STANLEY MEDICAL COLLEGE &HOSPITAL

CHENNAI- TAMIL NADU

APRIL 2012

CERTIFICATE

This is to certify that this dissertation entitled **"THE BACTERIOLOGY OF PLEURAL SPACE INFECTION AND CLINICAL, LABORATORY, AND PHYSICAL DETERMINANTS OF OUTCOME OF INFECTION"** submitted by **Dr.ARIVUDAINAMBI V.P** to the Tamil Nadu Dr.M.G.R Medical University is in partial fulfillment of the requirement of the award of **M.D. DEGREE BRANCH -XVI1(PULMONARY MEDICINE)** and is a bonafide research work carried out by him under direct supervision and guidance.

Signature of Unit Chief **Signature of the Professor and HOD**

Signature of the Dean

DECLARATION

I solemnly declare that the dissertation entitled **"THE BACTERIOLOGY OF PLEURAL SPACE INFECTION AND CLINICAL, LABORATORY, AND PHYSICAL DETERMINANTS OF OUTCOME OF INFECTION"** was done by me at the Government Stanley Medical College and Hospital during 2009- 2011 under the guidance and supervision of **PROFESSOR Dr.CHANDRASEKAR .C M.D.DTCD**. The dissertation is submitted to the Tamil Nadu Dr.MGR Medical University towards the partial fulfillment of requirements for the award of **M.D.DEGREE (BRANCH – XVI)** in Pulmonary medicine.

Place: Chennai.

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ACKNOWLEDGEMENT

I owe my thanks to the Dean,Government Stanley Medical College and Hospital, Professor **Dr.S.GEETHA LAKHSMI M.D PhD,** for allowing me to avail the facilities needed for my dissertation work.

My beloved Head of the Department of Pulmonary Medicine, **PROF. C. CHANDRASEKAR M.D. DTCD** has always guided me, by example and valuable words of advice and has always given me his moral support and encouragement throughout the conduct of the study and also during my post graduate course. I owe my sincere thanks to him.

I have great pleasure in expressing my deep sense of gratitude and respect for Associate Professors **DR.O.R.KRISHNARAJASEKAR MD DTRD, DR.R.SRIDAR MD DTRD, DR.MAHILMARAN MD DTRD.** Stanley Medical College and Hospital, Chennai, I thank my professors and mentors and guide for giving suggestions and guidance in preparing this dissertation.

I offer my heartfelt thanks to my Assistant Professor **Dr.Suriakumar M.D** (Microbiology), **DR. RAVICHANDRAN M.D,**& **DR.RAJA M.D** for his constant encouragement, timely help and critical

suggestions throughout the study and also for making my stay in the unit both informative and pleasurable.

My family and friends have stood by me during my times of need. Their help and support have been invaluable to this study. My patients, who form the most integral part of the work, were always kind and cooperative. I cannot but pray for their speedy recovery and place this study as a tribute to them and to the numerous others likely affected. Last but not the least I would like to sincerely thank all my fellow post graduate students for sharing their knowledge

ABBREVIATION AND ACRONYM

ADA= adenosine deaminase assay.

ANA= antinuclear antibody.

BNP= brain natriuretric peptide.

CPE= complicated parapneumonic effusion.

F= French (tube size).

Il= interleukin

Pco_2= partial pressure of carbondioxide.

PF= pleural fluid.

Po_2 = Partial pressure of oxygen.

WBC= white blood cells.

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INTRODUCTION

Pleural infection is a clinical problem with annual incidence of up to 80,000 cases in the western world. Mortality and morbidity is high; 20% of patients with empyema die and approximately 20% require surgery to recover within 12 months of their infection.[1 2]

Despite the advent of effective antibiotics, bacterial pneumonia still results in significant morbidity and mortality in the population. In one study of 1,424 patients hospitalized with community-acquired pneumonia, patients with pleural effusions were 2.7 times more likely to be treatment failures than were those without pleural effusions.[3]

In another study, the relative risk of mortality in patients with community-acquired pneumonia was 7.0 times higher for patients with bilateral pleural effusions and 3.4 times higher for patients with unilateral pleural effusion of moderate or greater size as compared with other patients with community-acquired pneumonia alone [4].

Most pleural effusions associated with pneumonia resolve without any specific therapy directed toward the pleural fluid, but approximately 10% of patients require operative intervention. Delay in instituting proper

therapy for these effusions is responsible for some of the morbidity associated with parapneumonic effusions. [5]

For a simple parapneumonic effusion antibiotics according to culture and sensitivity will suffice.

In complicated parapneumonic effusion and empyema intervention in the form of thoracentesis, tube thoracostomy, thoracoscopic intervention, surgery will be needed accordingly.

AIM OF THE STUDY

To describe the bacteriology of pleural space infection and to determine the prognostic factors implied in the outcome of pleural space infection chiefly simple complicated and complex parapneumonic effusion admitted from period of June 2009 to June 2011 in tertiary care respiratory institute (Government hospital of thoracic medicine Tambaram sanatorium) under Stanley medical college Chennai.

It was hypothesized that there could be clinical , laboratory and biochemical prognostic factors determining the outcome of parapneumonic effusion that if identified at the optimal period would result in reduction in morbidity and mortality associated with parapneumonic effusion. This study was aimed at identification of such determinants.

LITERATURE REVIEW

Anatomy of the Pleura

The pleural space has two membranes, the visceral pleura covering the lung and the parietal pleura covering the chest wall and diaphragm. Into pleural space, normal protein and liquid enter from circulation and are eliminated by the parietal pleural lymphatic. Pleural pressure is sub atmospheric and ensures inflation of the lung. The mesothelium is leaky, excess fluid migrate across into this lower-pressure, high-capacitance space and collect as a pleural effusion. Each pleural membrane has a single cell layer of mesothelial cells. There shape may be flat to cuboidal or columnar, based on the degree of stretching of the underlying submesothelial tissue. These most numerous cell of the pleural space, may have a variety of functions important to pleural biology[7] .Mesothelial cells can secrete the macromolecular components of the extracellular matrix and organize them into mature matrix, phagocytose particles, and secrete neutrophil and monocyte chemotactic factors that may be important for inflammatory cell recruitment into the pleural spaces can produce fibrinolytic and procoagulant factors. The mesothelial cells

produce cytokines such as transforming growth factor-β, epidermal growth factor (EGF), and platelet-derived growth factor, cytokines that are important in pleural inflammation and fibrosis.

Mesothelial cells produce hyaluronan but not mucin, express keratin microfilaments, stain negatively with epithelial-specific antibodies (Ber-EP4, B72.3, Leu.M1, and CEA), and stain positively for calretinin and mesothelin—all features that are important for histochemical and immunohistochemical identification of the cells in pleural effusions.[8 9]

Physiology of pleural space

Normal pleural fluid arises from the systemic pleural vessels in both pleurae, flows across the leaky pleural membranes into the pleural space, and exits the pleural space via the parietal pleural lymphatic.[10,11]

The pleural space is similar to other interstitial spaces of the body. The evidence for this view.

1. Intrapleural pressure is lower than the interstitial pressure of either of the pleural tissues. Pressure gradient makes a liquid movement into but not out of the pleural space.

2. The pleural membranes are leaky to liquid and protein. Whether tested in vitro[12,13] or in situ,[14] the pleura offers little resistance to liquid or protein movement.

3. Various transporters and aquaporins are present in mesothelial cells but no role in reabsorption of effusions.[15] Normal pleural liquid has been reported to be alkaline with higher bicarbonate than plasma, No evidence for active mesothelial role in generation of bicarbonate gradient, which can more likely be explained by a passive response to protein gradients (Donnan equlibrium).[16]

4. Pleural liquid entry is compatible with known interstitial flow rates as it is slow. Noninvasive studies of the equilibration of radiolabeled albumin have shown that, the rate of entry pleural liquid is approximately about 0.5 mL/hr or 12 mL/day in an adult human and 0.01 mL/kg/hr in a sheep, .[17]

5. The protein concentration of normal pleural liquid is low in sheep & humans[11] impling sieving of the protein across a high-pressure gradient. The protein concentration of sheep pleural liquid (10 g/L) and pleural–to–plasma protein concentration ratio (0.15) are similar to those of filtrates from high-pressure systemic vessels. By comparison, a filtrate from low-pressure pulmonary vessels has a higher protein concentration (45 g/L) and ratio (lymph–to–plasma protein concentration ratio 0.69.[19]

6. Pleural fluid exits by bulk flow, not by diffusion or active transport, evident by the protein concentration of pleural effusions remaining constant as the effusion is absorbed, as is with bulk flow. Absorption by diffusion or active transport, proteins would diffuse at a slower rate, and the protein concentration would progressively increase. Moreover erythrocytes instilled into the pleural space are absorbed intact and in almost the same proportion as the liquid and protein.[11] This denotes that the major route of exit is via holes large enough to accommodate sheep erythrocytes (6–8 mm diameter). The only possible exit is via the parietal pleural stomata (10–12 mm diameter) into the pleural lymphatics.[11]

Pathophysiology

To form an effusion, it is likely that *both* the entry rate of liquid must increase and the exit rate must decrease. If only the entry rate increased, it would require rate more than 30 times normal to exceed the reserve lymphatic removal capacity; if the exit rate decreased, it would take more than a month at the normal entry rate of 12 mL/day to produce an effusion detectable by chest radiograph. [19]

Most of pleural infection exemplify a progressive process that metamorphoses' a 'simple' self resolving parapneumonic pleural

effusion into a ‘complicated’ multiloculated fibrinopurulent collection associated with clinical and/or biochemical features of sepsis . [20]

The development of empyema in association with pneumonia has been classified into three stages as:

(1) a simple exudate,

(2) a fibrinopurulent stage and

(3) a later organising stage with scar tissue (pleural peel) formation. [20]

In the early exudative stage there is fluid movement into the pleural space due to increased capillary vascular permeability. There is also production of proinflammatory cytokines such as interleukin 8 (IL-8) and tumor necrosis factor alfa (TNFa).[21] Mesothelial cells to facilitate fluid entry . At first, the fluid is a free-flowing exudate characterised by a low white cell count, an LDH level less than half that in the serum, normal pH and glucose levels and does not contain bacterial organisms. [22-26] This stage, when the pleural fluid is a straightforward sterile exudate, is often called a ‘simple parapneumonic effusion’. Treatment with antibiotics at this stage is likely to be adequate and most effusions of this type do not require chest tube drainage. [23- 24]

If optimal treatment is not commenced, a simple parapneumonic effusion may progress to the fibrinopurulent stage with increasing fluid accumulation and bacterial invasion across the damaged endothelium. Bacterial invasion accelerates the immune response, promoting further migration of neutrophil and activation of the coagulation cascade leading to increased procoagulant and depressed fibrinolytic activity. [27-28]

The stages of a parapneumonic effusion are

(1) exudative (capillary leak) - period of 5 to 7 days;

(2) fibrinopurulent or bacterial invasion and fibrin formation stage, - after 7 days up to 2 weeks; and

(3) the organizational or empyema stage, which generally occurs within 2 to 4 weeks of onset of the pleural effusion.

An anaerobic infection, prolonged pneumonia history, failure to respond to antibiotic therapy, virulence of the underlying bacterial pathogen, and hypoalbuminemia also suggest the presence of a complicated parapneumonic effusion.[29-30]

Bacteriology of community-acquired and hospital-acquired pleural infection [2]

Common organisms

Community-acquired Streptococcus spp. (52%)

- S milleri
- S pneumoniae
- S intermedius
- Staphylococcus aureus (11%)

Gram-negative aerobes (9%)

- Enterobacteriaceae
- Escherichia coli

Anaerobes (20%)

- Fusobacterium spp.
- Bacteroides spp.
- Peptostreptococcus spp.
- Mixed

Hospital-acquired Staphylococci

- Methicillin-resistant S aureus (MRSA) (25%)
- S aureus (10%)

Gram-negative aerobes (17%)

- E coli
- Pseudomonas aeruginosa
- Klebsiella spp.
- Anaerobes (8%)

Transudate vs Exudate

Light and colleagues[31] have devised a diagnostic rule for pleural effusion as an exudate. An exudative effusion if any one of : (1) a PF lactate dehydrogenase (LDH) level >0.67, *ie*, the upper limit of normal for the laboratory serum LDH value; (2) a PF/serum protein ratio > 0.5; and (3) a PF/serum LDH ratio >0.6. The use of a three-test combination with "and/or" rule maximizes diagnostic sensitivity for detecting exudative pleural effusions but lowers specificity.

In a receiver operating characteristic analysis of 200 consecutive patients with pleural effusions, a PF LDH of 163 IU/L (serum upper limits of normal, 200; ratio _ 0.82) was the best cutoff point for an exudates (area under the curve, 0.89), followed by PF/serum total protein ratio of 0.5 (area under the curve, 0.86).

APPROACH TO PATIENTS WITH PLEURAL EFFUSION

Dullness to percussion, decreased breath sounds, egophony at the upper level of the effusion and decreased tactile fremitus are the physical findings of a pleural effusion. Signs can include asymmetrical chest expansion or even bulging of the intercostal spaces with large effusions. The most useful physical findings were dullness to percussion and decreased tactile fremitus.[33]

Abnormal chest radiograph should arise suspicion. Increased densities attributed to parenchymal infiltrates when they can represent pleural fluid. The posterior costophrenic sulcus is the most dependent part of the thoracic cavity where free pleural fluid gravitates to when the patient is upright. Therefore, if the posterior costophrenic angle is blunted or if the posterior part of the diaphragm is not visible on the lateral chest radiograph bilateral decubitus chest radiographs or an ultrasonic examination of the pleural space should be obtained to ascertain whether free pleural fluid is present. If the distance between the inside of the thoracic cavity and the outside of the lung is less than 10 mm, the pleural

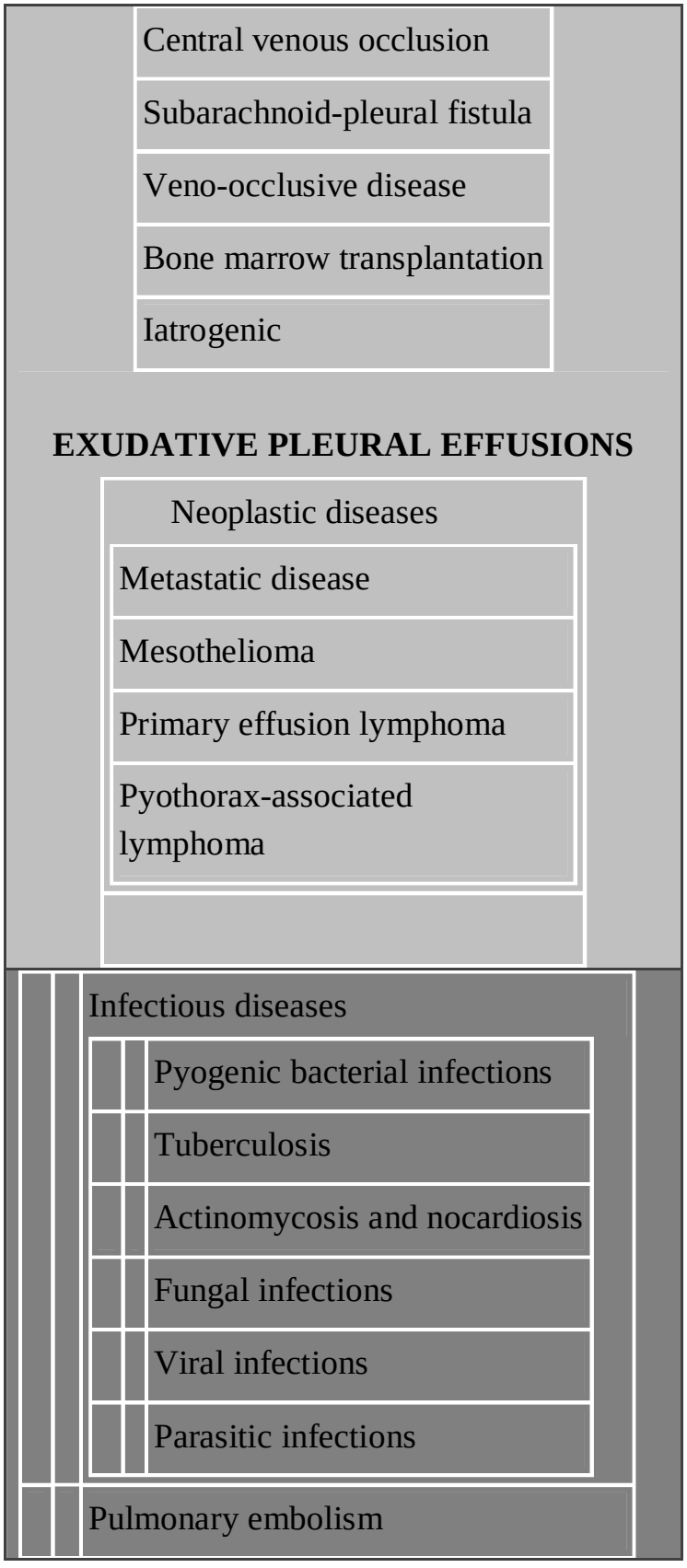
effusion is not likely to be clinically significant and in any case will be difficult to obtain by thoracentesis. If the distance is greater than 10 mm, an effort should be made to determine the cause of the pleural effusion.

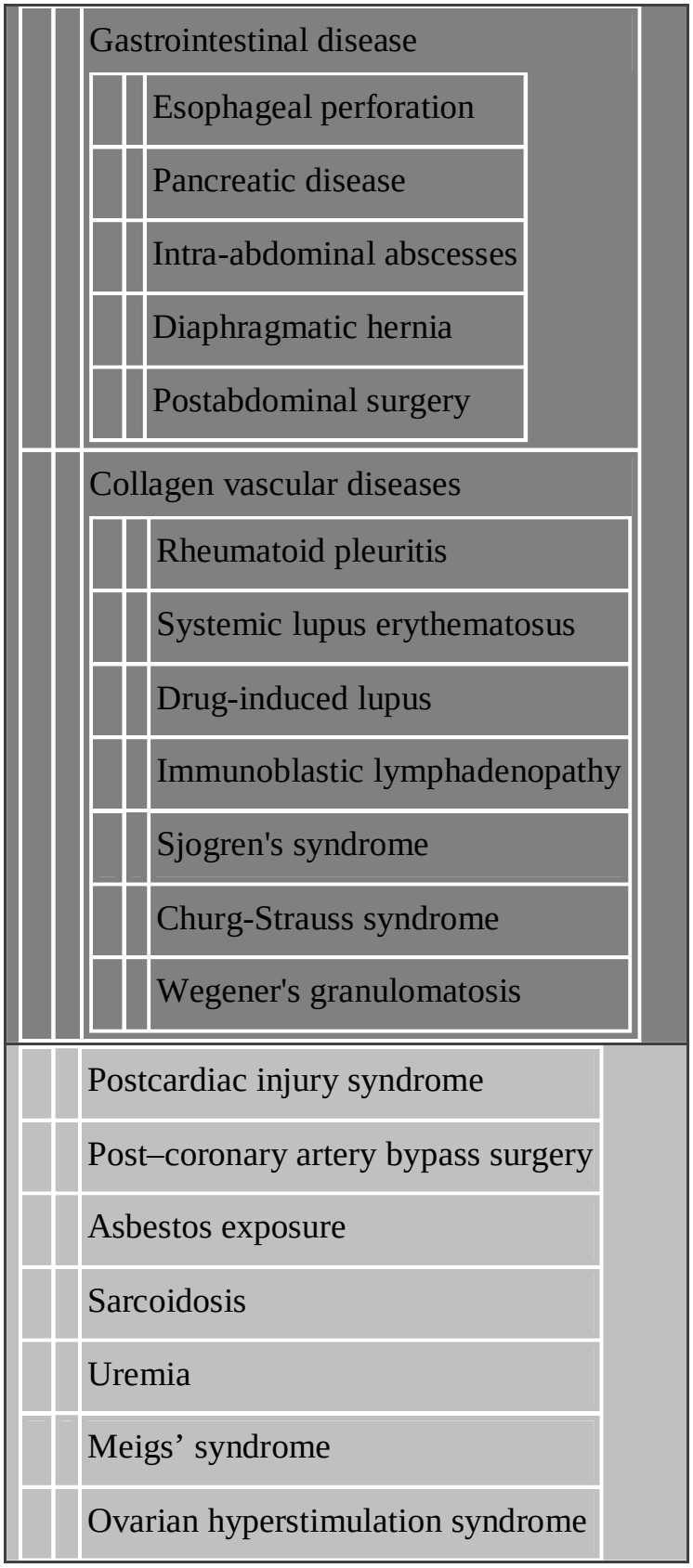
Differential Diagnosis of Pleural Effusion

Pleural effusion accompanies many different diseases. Most common causes of pleural effusions is in table. Almost all transudative pleural effusions are due to CHF and cirrhosis. Pneumonia, malignant pleural disease, pulmonary embolism, and gastrointestinal disease account for at least 90% of all exudative pleural effusions.

TABLE -- Differential Diagnoses of Pleural Effusions [34]

TRANSUDATIVE PLEURAL EFFUSIONS
Congestive heart failure
Pericardial disease
Hepatic hydrothorax
Nephrotic syndrome
Peritoneal dialysis
Urinothorax
Myxedema
Fontan procedure


Central venous occlusion
Subarachnoid-pleural fistula
Veno-occlusive disease
Bone marrow transplantation
Iatrogenic
EXUDATIVE PLEURAL EFFUSIONS
Neoplastic diseases
Metastatic disease
Mesothelioma
Primary effusion lymphoma
Pyothorax-associated lymphoma
Infectious diseases
Pyogenic bacterial infections
Tuberculosis
Actinomycosis and nocardiosis
Fungal infections
Viral infections
Parasitic infections
Pulmonary embolism


Gastrointestinal disease
Esophageal perforation
Pancreatic disease
Intra-abdominal abscesses
Diaphragmatic hernia
Postabdominal surgery
Collagen vascular diseases
Rheumatoid pleuritis
Systemic lupus erythematosus
Drug-induced lupus
Immunoblastic lymphadenopathy
Sjogren's syndrome
Churg-Strauss syndrome
Wegener's granulomatosis
Postcardiac injury syndrome
Post–coronary artery bypass surgery
Asbestos exposure
Sarcoidosis
Uremia
Meigs’ syndrome
Ovarian hyperstimulation syndrome

- Yellow nail syndrome
- Drug-induced pleural disease
  - Nitrofurantoin
  - Dantrolene
  - Methysergide
  - Bromocriptine
  - Procarbazine
  - Amiodarone
  - Dasatinib
- Radiation therapy
- Electric burns
- Iatrogenic injury
- Hemothorax
- Chylothorax

Separation of Exudates from Transudates

On nearly every patient with free pleural fluid that measures more than 10 mm on the decubitus radiograph a diagnostic thoracentesis should be performed. If CHF is obvious, postpone the thoracentesis until the heart failure is treated. If the patient is febrile or has pleuritic chest pain

or if the effusions are not of comparable size on both sides, a thoracentesis should be performed without delay.

Thoracentesis when performed by an experienced operator is a safe procedure.

It can be safely performed in patients with coagulopathies and thrombocytopenia and in patients on positive mechanical ventilation because of the small-bore needle required.[35]

Exudative pleural effusions meet at least one of the following criteria, whereas transudative pleural effusions meet none[32]: (1) pleural fluid protein–to–serum protein greater than 0.50; (2) pleural fluid LDH–to–serum LDH greater than 0.60; and (3) pleural fluid LDH greater than two thirds of the upper normal limit for serum. If none of these criteria is met, the patient has a transudative pleural effusion, and the pleural surfaces can be ignored while the CHF, cirrhosis, or nephrosis is treated. In the rare cases in which malignancy has been associated with a transudate, extrapleural effects of the tumor or other causes such as concurrent CHF are the most likely cause as evidenced by the rarity of a positive cytology in those effusions.[19 36]

The previously discussed criteria may misidentify a transudative effusion as an exudative effusion in as many as 25% of cases. If a patient appears to have a transudative effusion clinically, additional tests can be assessed to verify its transudative etiology. If the difference between the protein concentration of serum and the pleura exceeds 3.1 gm/dL, the patient in all probability has a transudative effusion.[37] If pleural concentrations of N-terminal brain natriuretic peptide (NT-BNP) are elevated (>1300 pg/mL), the patient likely has a transudate from a cardiac cause.[38]

Differentiating Exudative Pleural Effusions

Pneumonia, malignancy, and pulmonary embolism account for the great majority of all exudative pleural effusions. Undiagnosed exudative pleural effusions, the appearance of the fluid should be noted, pleural fluid protein and LDH levels (if not already measured), glucose level, differential cell count, and microbiologic and cytologic studies should be obtained.[39]

In selected patients, other tests on the pleural fluid, such as pH, amylase level, antinuclear antibody (ANA) level, rheumatoid factor level, adenosine deaminase (ADA), lipid analysis, and so forth, may be of value.

Appearance of Pleural Fluid

The gross appearance and odor of the pleural fluid should be noted. If pleural fluid smells putrid, the patient has a bacterial infection (probably anaerobic). If smells like urine, patient has a urinothorax. If the pleural fluid is bloody, a pleural fluid hematocrit should be obtained. If it is greater than 50% that of the peripheral blood, the patient has a hemothorax and inserting chest tubes the physician should give strong consideration. If the pleural fluid hematocrit is less than 1%, the blood in the pleural fluid has no clinical significance. If the pleural fluid hematocrit is between 1% and 50%, the patient most likely has malignant pleural disease, a pulmonary embolus, or a traumatically induced pleural effusion.[40]

The supernatant of the pleural fluid should be examined if the pleural fluid is turbid, milky, or bloody. If turbidity clears with centrifugation, the turbidity is due to cells or debris in the pleural fluid. The patient probably has a chylothorax or a pseudochylothorax if the turbidity persists after centrifugation. They can be differentiated by the patient's history, examination of the sediment for cholesterol crystals, and lipid analysis of the supernatant. The disease process is acute, no thickened pleural surfaces, no cholesterol crystals present, and the

pleural fluid triglyceride level is usually above 110 mg/dL (1.24 mmol/L) with chylothorax)., The disease process is usually chronic, the pleural surfaces are usually thickened, there may be cholesterol crystals, and the pleural fluid triglyceride level is usually not elevated with pseudochylothorax.

Pleural Fluid Protein

If the protein level is above 5.0 g/dL, the likelihood of the diagnosis of tuberculous pleurisy is increased, the patient probably has a urinothorax, an effusion secondary to peritoneal dialysis, a leak of CSF into the pleural space, or an effusion secondary to the misplacement of a central intravascular line iff the pleural fluid protein level is *very* low (<0.5 g/dL).

Pleural Fluid Lactate Dehydrogenase

LDH concentration should be measured during diagnostic thoracentesis because the level of LDH in the pleural fluid reflects the degree of inflammation in the pleural space. LDH concentration increases or decrease with serial thoracentesis, is directly proportional the degree of inflammation in the pleural space.[41]

Pleural Fluid Glucose

A thickened, infiltrated pleura leading to an impaired diffusion of glucose into the pleural space plus increased metabolic activity leading to increased glucose utilization within the pleural space can result in a low glucose concentration. In all undiagnosed exudative pleural effusions the demonstration of a reduced pleural fluid glucose level (<60 mg/dL, 3.33 mmol/L) points to possibilities of parapneumonic effusion, malignant effusion, tuberculous effusion, rheumatoid effusion, hemothorax, paragonimiasis, or the Churg-Strauss syndrome.[41] Tube thoracostomy should be considered if a patient with a parapneumonic effusion has a pleural fluid glucose level below 40 mg/dL (2.22 mmol/L). Rheumatoid pleural effusions have a pleural fluid glucose level below 30 mg/dL (1.66 mmol/L).[42] Patients with pleural effusion secondary to systemic lupus erythematosus (SLE) will have a pleural fluid glucose level above 80 mg/dL (4.44 mmol/L).[43] Patients with malignant pleural disease and a low pleural fluid glucose level usually have a positive pleural fluid cytology.[44]

Pleural Fluid White Cell Count and Differential

The cell count has been reported to be 1700 cells/mm^3.[45] In effusions, the cell count has limited diagnostic value. A pleural fluid white blood cell count of 1000/mm^3 roughly separates transudative from exudative pleural effusion.

A pleural fluid white blood cell count above 10,000/mm^3 is most common with empyemas and parapneumonic effusions, but is also seen with collagen vascular diseases, pancreatitis, pulmonary embolism, malignancy and tuberculosis.[40]

The differential cell count on the pleural fluid is much more useful. Macrophages (75%) followed by lymphocytes (23%) is the normal pleural space content.[45] For the pleural fluid differential cell count, the cells should be partitioned into the following categories: polymorphonuclear leukocytes, eosinophils, small lymphocytes, mesothelial cells, and other mononuclear cells. Pleural effusions due to an acute disease process such as pneumonia, pulmonary embolization, pancreatitis, intra-abdominal abscess, or early tuberculosis contain predominantly polymorphonuclear leukocytes. Pleural effusions due to a chronic disease process contain predominantly mononuclear cells.

Pleural fluid eosinophilia (≥10% eosinophils by differential count) is most commonly due to air or blood in the pleural space. The pleural liquid IL-5 levels correlates with the number and percentage of eosinophils in the pleural space.[46] Occasionally, no pleural fluid eosinophils are found in the initial thoracentesis, but many eosinophils are seen in a subsequent thoracentesis most likely due to entry of air or blood caused by the initial thoracentesis.[47] With traumatic hemothorax, pleural fluid eosinophilia does not occur until the second week. The eosinophilia appears to be due to production of IL-5 by $CD4^+$ T cells within the pleural space.[48] At times, the pleural fluid eosinophilia associated with a hemothorax can lead to eosinophilia in the peripheral blood.[49] The bloody pleural effusion complicating pulmonary embolism frequently contains many eosinophils.[50] With pneumothorax, pleural eosinophilia appears within 3 days of the pneumothorax and reaches a peak after 6 days.[51]

If the etiology of the eosinophilia is not evident, several unusual diagnoses should be considered. Eosinophilic cell count is seen with benign asbestos pleural effusions.[47] Patients with pleural effusions secondary to drug reactions (nitrofurantoin or dantrolene) typically have pleural fluid eosinophilia.[41] Typically eosinophilic with low glucose, low

pH, and high LDH level is the pleural fluid of patients with pleural paragonimiasis.[52] The Churg-Strauss syndrome is the only other disease that produces this constellation of pleural fluid findings.[53]

Mesothelial cells line the pleural cavities. It is unusual to find mesothelial cells in effusions due to tuberculosis. However, the absence of mesothelial cells is also common with other conditions in which the pleura becomes coated with fibrin, such as a complicated parapneumonic effusion.

Small lymphocytes, when accounting for more than 50% of the white blood cells in an exudative pleural effusion, indicate that the patient probably has a malignant or a tuberculous pleural effusion.[40 54] Because these two diseases can be diagnosed with needle biopsy of the pleura, the presence of pleural fluid lymphocytosis should alert the physician to consider needle biopsy of the pleura for diagnosis. Because most lymphocytic effusions contain a predominance of T cells ($CD4^+$) whether the diagnosis is malignancy or tuberculosis and hence separation of pleural lymphocytes into T and B lymphocytes has not been useful diagnostically.[55] A diagnosis of chronic lymphocytic leukemia or lymphoma is suspected. When, the pleural lymphocytes are predominantly of B-cell origin.[56]

Pleural Fluid Cytology

In up to 60% of the effusions caused by pleural malignancy the first pleural fluid cytologic study is positive for malignant cells.[40] 90% of effusions due to pleural malignancy have positive cytopathology if three separate specimens are analysed. Less than 25% of patients with Hodgkin's disease have positive cytology[58] whereas most patients with adenocarcinomas have positive cytology.[57] hence frequency of positive pleural fluid cytologic tests is dependent on the tumor type. During thoracoscopy, pleural lavage has been found to increase the diagnostic yield, perhaps by harvesting more fresh cells for analysis.[60] The percentage of positive diagnoses is obviously dependent on the skill of the cytologist. Immunohistochemical stains of malignant cells are used to confirm a diagnosis and to specify tumor type, with many new markers recently available.[8 59]

Other Diagnostic Tests for Malignancy

Abnormal numbers of specific chromosomes (aneuploidy) can be confirmed by fluorescent in situ hybridization (FISH) with chromosome-specific probes, thereby confirming that abnormal cells are indeed malignant.[61] DNA methylation is an early findings of malignancy can be

detected by methylation-specific polymerase chain reaction (PCR),[62] and gene expression patterns can help distinguish mesothelioma and adenocarcinoma.[63] EGF-receptor mutations can predict response to EGF-receptor tyrosine kinase inhibitors. Conversely, biomarkers have generally been disappointing due to nonspecificity.

Culture and Bacteriologic Stains

Pleural fluid from patients with undiagnosed exudative pleural effusions should be cultured for bacteria (both aerobically and anerobically), mycobacteria, and fungi. Gram's stain should also be obtained. In the case of a probable complicated parapneumonic effusion with an initial negative Gram's stain, the sediment of the pleural fluid should be stained because the bacteria will be precipitated in the sediment along with the white blood cells and the debris.

Amplification and sequencing of bacterial 16S ribosomal RNA has identified bacteria in pleural empyema, showing in one study that the bacteriology of pleural infections differed from that of pneumonia[64]

Other Diagnostic Tests for Pleural Fluid

Pleural Fluid pH and PCO_2

(1) Complicated parapneumonic effusion, (2) hemothorax (3) rheumatoid pleuritis, (4) tuberculous pleuritis, (5) urinothorax (6) esophageal rupture, (7) systemic acidosis, (8) paragonimiasis, (9) lupus pleuritis, or (10) malignant pleural disease are the pleural fluid pH can be reduced to less than 7.20[41]. The decreased pleural fluid pH appears to result from lactic acid and carbon dioxide accumulation in the pleural fluid.[65] Whether chest tubes should be inserted in patients with parapneumonic effusions is determined by the pleural fluid pH .[66]

Blood gas machine is ideal for the pH measurement; a pH meter or indicator paper is not useful for accurate measure.[67] Pleural fluid pH, altered by residual air or lidocaine in the syringe.[68]

Pleural Fluid Amylase

Esophageal perforation, pancreatic disease, or malignant disease are associated with pleural fluid amylase. The pleural fluid amylase concentration is elevated within 2 hours of esophageal rupture, the origin of the amylase is the salivary glands.[70,71] In effusions due to pancreaticopleural fistulas, the amylase concentration is extremely high

(>4000 IU/mL), reflective of the concentrations in pancreatic secretions.[72] In approximately 10% of malignant effusions, the pleural fluid amylase level is mildly elevated. The site of the primary tumor in such patients is usually not the pancreas.[73] Malignancy can be differentiated from pancreatic disease with amylase isoenzymes because the amylase with malignant effusions is primarily of the salivary type.[74]

CLASSIFICATION

Class 1 Non significant pleural effusion	<10 mm thick on decubitus x-ray. No thoracentesis
Class 2 Typical parapneumonic pleural effusion	>10 mm thick
	Glucose >40 mg/dL, pH >7.2
	LDH <3 Ã— upper limit normal for serum
	Gram's stain and culture negative
	Antibiotics alone
Class 3 Borderline complicated pleural effusion	7.0 <pH <7.20 and or
	LDH >3 Ã— upper limit normal and glucose >40 mg/dL
	Gram's stain and culture negative
	Antibiotics plus serial thoracentesis
Class 4 Simple complicated pleural effusion	pH <7.0 or glucose <40 mg/dL or
	Gram's stain or culture positive
	Not loculated not frank pus
	Tube thoracostomy plus antibiotics
Class 5 Complex complicated pleural effusion	pH <7.0 and/or glucose <40 mg/dL or
	Gram's stain or culture positive
	Multiloculated
	Tube thoracostomy plus fibrinolytics (rarely require thoracoscopy or decortication)
Class 6 Simple empyema	Frank pus present
	Single locule or free flowing
	Tube thoracostomy Â± decortications
Class 7 Complex empyema	Frank pus present
	Multiple locules
	Tube thoracostomy ± fibrinolytics
	Often require thoracoscopy or decortication

Classification of the American College of Chest Physicians

ACCP classification of parapneumonic effusions on the basis of the anatomical characteristics of the fluid (A), the bacteriology of the pleural fluid (B) and the chemistries (C) of the pleural fluid [75].

The anatomy (A) of the pleural effusion is based on the size of the effusion, whether it is free flowing and whether the parietal pleural is thickened.

A_0 effusions - Small effusions (<10 mm in thickness on the decubitus radiographs, ultrasound examination, or CT scans) & free flowing.

A_1 effusions - > 10 mm in thickness but occupy less than 50% of the hemithorax, are free flowing, no parietal pleural thickening.

A_2 effusions occupy more than 50% of the hemithorax or are loculated and/or are associated with thickening of the parietal pleura.

The bacteriology (B) of the effusion is based on whether smears or cultures are positive.

B_x effusions - culture and Gram's stain results are unknown, because the effusion was small and a thoracentesis was not done.

B_0 effusions - negative Gram's stains and cultures of the pleural fluid.

B_1 effusions- Gram's stain or culture are positive, but the pleural fluid is not pus.

B_2 effusions -pleural fluid is pus.

The chemistry (C) of the effusion is based on the pH of the pleural fluid.

C_x effusions - pleural fluid pH is unknown (thoracentesis was not done).

C_0 effusions - pleural fluid pH greater than 7.20.

C_1 effusions- pleural fluid pH less than 7.20.

For accurate pleural fluid pH, the pleural fluid must be measured with a blood gas machine 74.

On the basis of the A, B, and C classification, the effusion is categorized.

Category 1 effusion- Small (<10 mm on decubitus, CT scan or ultrasound)

Free-flowing.

Effusion is small, no thoracentesis

Bacteriology and chemistry - unknown.

Risk of a poor outcome very low.

Category 2 effusion - Small to moderate in size (>10 mm & <1/2 of hemithorax)

Free flowing.

Gram's stain and culture of the pleural fluid are negative

Pleural fluid pH > 7.20.

The risk of a poor outcome effusion is low.

Category 3 effusion one of the criteria

(a) effusion > 1/2 the hemithorax, is loculated, or with a thickened parietal pleura;

(b) Gram's stain or culture - positive; or

(c) pleural fluid pH < 7.20 or

(d) pleural fluid glucose < 60 mg/dL.

Risk of a poor outcome - moderate.

Category 4 effusion - pleural fluid that is pus.

The risk of a poor outcome - High

Overall Treatment Plan

Treatment plan for parapneumonic effusions and empyema, are

- ❖ Diagnostic thoracentesis,
- ❖ Therapeutic thoracentesis,
- ❖ Tube thoracostomy,
- ❖ Tube thoracostomy with thrombolytics,
- ❖ Thoracoscopy, and
- ❖ Thoracotomy with decortication.

One of these treatments will be needed If the patient has any of the poor prognostic factors, It is essential not to continue any of the treatment that is not working for more than a day or so.[78 79]

The effectiveness of a given treatment is evaluated by the clinical status of the patient together with the amount and the characteristics of the pleural fluid. Success denotes that the patient appears to be responding, and amount of pleural fluid is not large, the characteristics of the fluid are improving.

More invasive procedures are not indicated for pleural thickening or loculation alone. Chest CT scans are very useful in evaluating the adequacy of drainage of the pleural space.

Diagnostic versus Therapeutic Thoracentesis

A thoracentesis should be performed to determine if the patient has any of the prognostic factors. The risks of a diagnostic and a therapeutic thoracentesis are comparable, and a therapeutic thoracentesis might prevent the need for further procedures, therefore a therapeutic thoracentesis is recommended.[78] After thoracentesis pleural fluid recurs, Poor prognostic findings in the initial pleural fluid indicate the need for more drainage—either an additional therapeutic thoracentesis or a tube thoracostomy. The absence of poor prognostic findings in a patient who is improving clinically indicates that additional drainage is not necessary. Three therapeutic thoracenteses are all that is recommended.[78]

If the pleural fluid is loculated, tube thoracostomy should be performed if any of the other poor prognostic factors are present. To insert a small pigtail catheter is an alternative to therapeutic thoracentesis.

Chest Tubes

Tube thoracostomy should be not be delayed, because a complicated parapneumonic effusion can progress from free-flowing pleural fluid to loculated pleural fluid within hours. Mortality can be attributed to a delay in obtaining adequate pleural drainage.[76] The chest tube should be positioned in the most dependent part of the effusion. On the optimal size of the chest tube for drainage ,there is no agreement.[77] Large (26–36 French) chest tubes were recommended in the past because of obstruction by the thick fluid. Smaller tubes under image or ultrasound guidance can suffice. The chest tube should be left in place until the volume of the pleural drainage per 24 hours is less than 50 mL and until the draining fluid becomes clear yellow. Closed-tube drainage of a complicated parapneumonic effusion is associated with improvement in the clinical and radiologic status of the patient within 24 to 48 hours is termed Successful. Either if the pleural drainage is unsatisfactory or the patient is receiving the wrong antibiotic there will be no significant improvement after this period. Then the culture results should be

reviewed and the adequacy of the pleural drainage should be assessed by imaging or ultrasound. Another chest tube should be inserted if the pleural space is inadequately drained, a fibrinolytic agent or saline can be injected intrapleurally or surgery can be performed. Poor positioning of the tube, loculated pleural fluid, obstruction of the chest tube or inadequate expansion of the underlying lung due to coating of the visceral pleura leads to inadequate drainage.

Intrapleural Thrombolytic Agents

Drainage of complicated parapneumonic effusions as a result of pleural loculations by fibrin membranes were difficult. Intrapleural fibrinolytics will degrade the fibrin membranes and facilitate drainage of the pleural fluid in theory. But most important study on the use of intrapleural fibrinolytics for the treatment of complicated parapneumonic effusion, even in subgroup analysis the administration of streptokinase was not beneficial.[80] In this multicenter, randomized, controlled, double-blind study, 454 patients were randomized to receive 250,000 IU streptokinase or saline at a total volume of 30 mL twice a day. There was no difference in mortality, the need for surgical intervention, or the length of hospitalization. At the present fibrinolytics are not recommended for routine use. Tissue plasminogen activator (t-PA) is an alternative

fibrinolytic agent.[81] A study in rabbits demonstrated that the combination of t-PA and recombinant DNase drained empyema fluid better than either agent alone.[82]

Thoracoscopy

If tube thoracostomy fails, thoracoscopy a more invasive thoracoscopic procedure is needed, the fibrin membranes making the loculations are broken down and removed, the peel covering the visceral pleural can sometimes be removed with thoracoscopy and, at the end a chest tube is inserted.[83] A chest CT will provide anatomic information about the size and the extent of the empyema cavity hence need prior to thoracoscopy.

A thickened visceral pleural peel without septations suggests that the empyema may be chronic and probably will not be compliant to thoracoscopic debridement alone.

Decortication

After tube thoracostomy and thoracoscopy fails this is the procedure of choice. A full thoracotomy is performed to remove all fibrous tissue and pus from the pleural space. The decortication is easier to perform with thoracotomy and hence offers advantage over

thoracoscopy . Pleural sepsis eliminated by decortication and it allows the underlying lung to expand. Decortication should not be performed for removing thickened pleura, as it resolves spontaneously over several months. Persistance of pleural thickening after 6 months, with reduction in pulmonary function sufficiently to limit the patient's activities, decortication should be option.

Open Drainage (Eloesser Flap)

Under local anesthesia, a skin flap overlying the lower part of the empyema collection is made with a U-shaped incision, to open the empyema cavity the ed rib segments and parietal pleura are excised; a semipermanent opening is made by suturing the skin flap inside the cavity into which one or more large-bore short tubes are inserted. This procedure allows complete drainage and frees the patient from attachment to chest-tube bottles.[83] The cavity should be irrigated daily with a mild antiseptic solution after the procedure, and a colostomy bag placed over the tubes can be used to collect the drainage from the tubes . In those patients who are too ill to tolerate the more extensive procedure Open drainage is preferred to decortications and can be used for a prolonged period. The median time for healing was over 120 days for drainage in one study of 53 patients treated by an open-drainage procedure.[84]

Bronchopleural Fistula Complicating Empyema

Adequate pleural drainage is crucial when an empyema is complicated by a bronchopleural fistula, If no drainage exteriorly with chest tubes it is likely to drain interiorly throughout the tracheobronchial tree, producing a severe diffuse pneumonia. Whenever the chest radiograph reveals an air-fluid level or the patient expectorates copious quantities of sputum while lying on one side (by gravity dependent drainage of the empyema into the bronchial tree) and not while lying on the other side a bronchopleural fistula should be suspected.

MATERIALS AND METHODS

The study is prospective analysis of patients cohort admitted to the tertiary care institute from the period of July 2009 to July 2010.

INCLUSION CRITERIA

(1) All patients with parapneumonic effusion admitted to the institute above age of fifteen years.

(2) All patients diagnosed to have simple parapneumonic effusion

(3) All Complicated parapneumonic effusion

(4) All Complex parapneumonic effusion.

EXCLUSION CRITERIA

(1) All patients with effusion of other causes like trauma, iatrogenic causes,were excluded.

(2) Paediatric parapneumonic effusion excluded.

(3) Tuberculous pleural effusion excluded.

Definitions

1. Parapneumonic effusion

Any pleural effusion associated with bacterial pneumonia, lung abscess, or bronchiectasis is a parapneumonic effusion [27].

2. Empyema: An empyema, by definition, is pus in the pleural space

Empyema as pleural fluid with a specific gravity greater than 1.018, a WBC count greater than 500 cells/mm^3, or a protein level greater than 2.5 g/dL- weese[29]. Vianna [30] defined an empyema as pleural fluid on which the bacterial cultures are positive or the WBC is greater than 15,000/mm^3 and the protein level is above 3.0 g/dL.

The term empyema - Pleural effusions with thick, purulent appearing pleural fluid.

Successful closed-tube drainage of a complicated parapneumonic effusion is associated with improvement in the clinical and radiologic status of the patient within 24 to 48 hours.

OUTCOME DEFINITION

Success : In complicated parapneumonic effusion it is defined as absolute drainage of the effusion or improvement in sepsis syndrome (fever & leucocytosis) after 2 weeks of appropriate antibiotics in accordance with culture and sensitivity.

Failure: Defined as incomplete drainage with failure of resolution of septic symptoms (fever, leucocytosis) or a fatal outcome.

Patients characteristics studied

Patients characteristics such as

(1) Age

(2) Sex

(3) Co-morbid illness like diabetes mellitus, alcoholic liver diseases

(4) Blood glucose

(5) Pleural fluid biochemical parameters such as P_H , glucose, Culture & staining characteristics, leukocyte count.

(6) Symptoms such as fever

(7) Other characteristics such as fibrin peel on thoracoscopy imaging evidence of loculation, serum protein(albumin) , total wbc count were taken up for analysis.

Factors Suggesting That a More Invasive Approach Will Be Necessary for the Resolution of a Parapneumonic Effusion*

1.	Pus is present in the pleural space.
2.	Positive pleural fluid Gram stain.
3.	Pleural fluid glucose < 60 mg/dL.
4.	Pleural fluid pH < 7.20.
5.	Positive pleural fluid culture.
6.	Pleural fluid lactate dehydrogenase > three times upper normal limit for serum.
7.	Loculated pleural fluid.

Chest tube were inserted fulfilling the above criteria by surgeon pulmonary physician. The size used were 28F, 32F.

P_H was measured using blood gas analyzer.

Hypoalbuminemia first appearing after 2 weeks of antibiotic was considered for analysis.

Statistical analysis

Bacteriology of the pleural infection defined and determinants (clinical, laboratory, & physical) of outcome in pleural space infection especially complicated & complex parapneumonic effusion were determined . Outcome were defined as success and failure.

Univariate analysis of the variables done

Comparison of means by independent student t test & chi-square test was done.

For dichotomous dependent variable confounding factors removed independent predictors determined using Logistic regression analysis especially binary logistic regression forward wald & backward conditional regression.

SPSS version 17 software was used to compute the results.

RESULTS

TABLES No -1

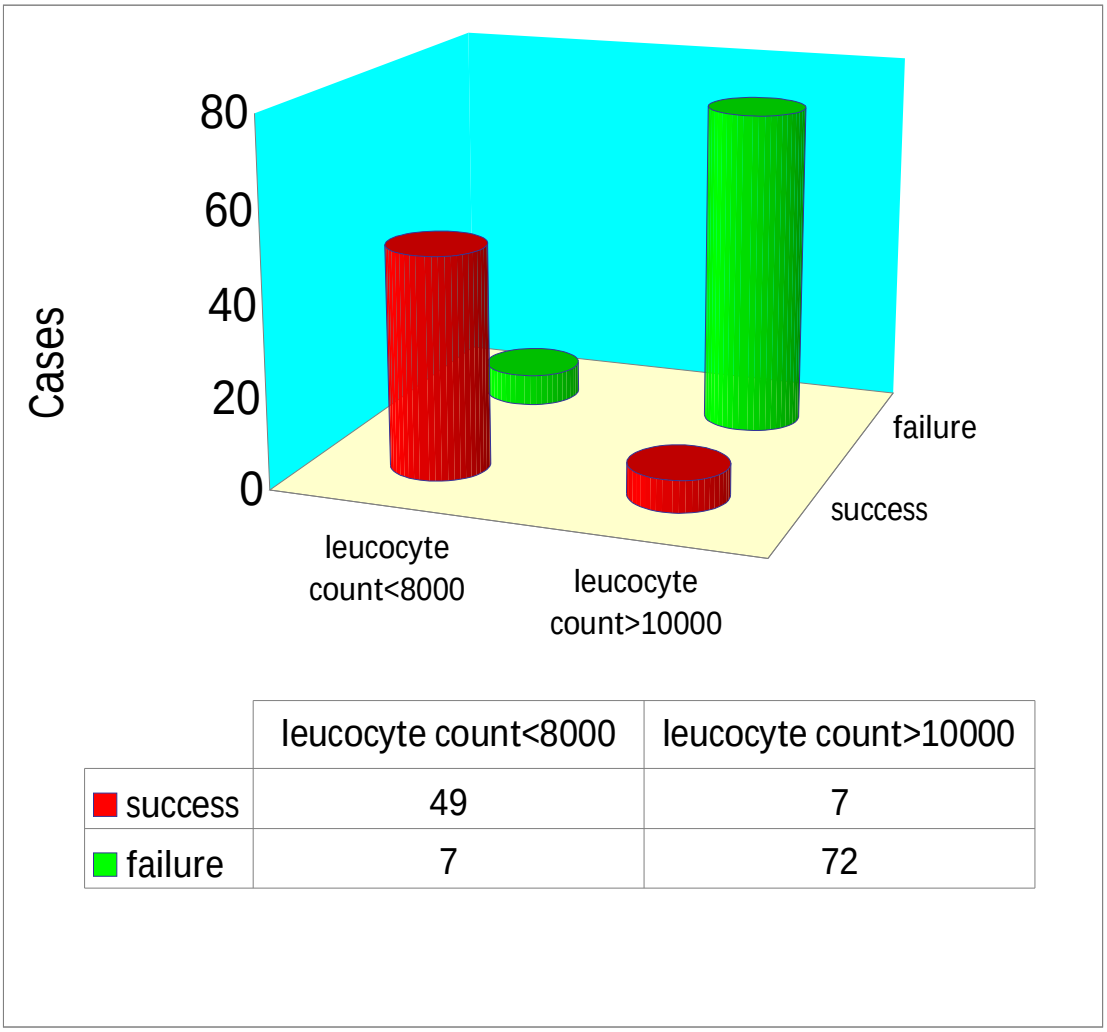
AGE				
Outcome	**Sex**	**Mean**	**N**	**Std. Deviation**
Failure	Male	47.1364	66	8.71583
	Female	46.1538	13	7.28979
	Total	46.9747	79	8.46255
success	Male	47.1250	40	10.98994
	Female	48.3750	16	7.63217
	Total	47.4821	56	10.09229
Total	Male	47.1321	106	9.58578
	Female	47.3793	29	7.43262
	Total	47.1852	135	9.14084

Cross table depicting mean age of male and female patients in success and failed outcome in various form of parapneumonic effusion

Tab-2 Crosstable depicting mean hydrogen ion concentration(P_H) of male and female patients in success and failed outcome in various form of parapneumonic effusion

Hion				
outcome	**Sex**	**Mean**	**N**	**Std. Deviation**
Failure	Male	5.679	66	1.3597
	Female	6.162	13	.9921
	Total	5.758	79	1.3132
Success	Male	7.495	40	.8171
	Female	7.750	16	1.0360
	Total	7.568	56	.8830
Total	Male	6.364	106	1.4748
	Female	7.038	29	1.2819
	Total	6.509	135	1.4576

GRAPHS

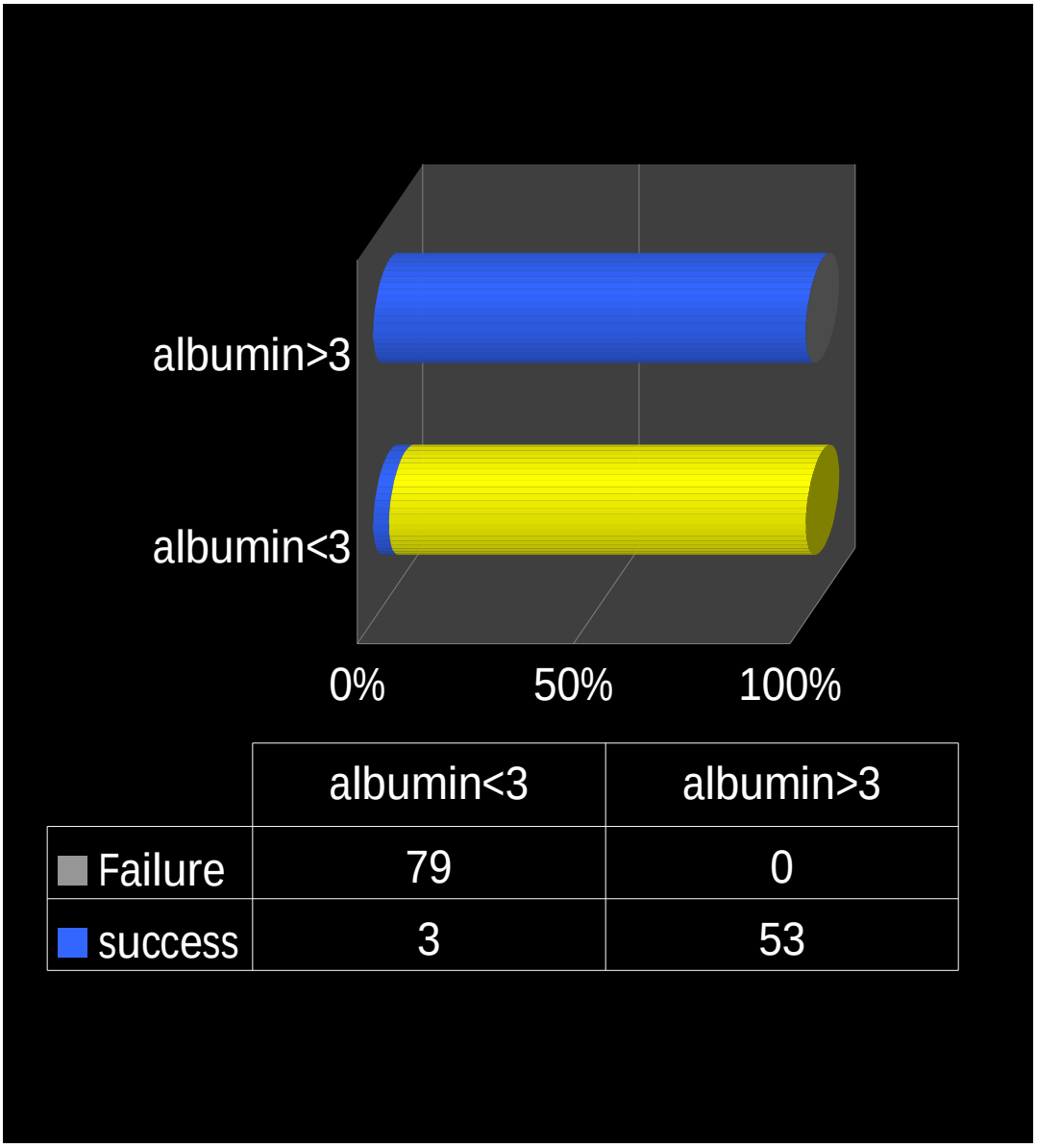


	leucocyte count<8000	leucocyte count>10000
success	49	7
failure	7	72

Graph depicting cross tabulation of success and failure with respesct to leucocyte count

Tab-3Cross table depicting mean white blood cell count in pleural fluid of male and female patients in success and failed outcome in various form of parapneumonic effusion

WBC				
Outcome	**Sex**	**Mean**	**N**	**Std. Deviation**
Failure	Male	12268.03	66	2857.609
	Female	9923.62	13	3886.823
	Total	11882.24	79	3145.546
success	Male	6706.55	40	2550.524
	Female	5655.56	16	1178.466
	Total	6406.27	56	2284.960
Total	Male	10169.36	106	3848.093
	Female	7568.83	29	3447.438
	Total	9610.73	135	3903.296



	albumin<3	albumin>3
Failure	79	0
success	3	53

Graph depicting cross tabulation of success and failure with respesct to Serum albumin

Tab -4 Cross table depicting mean serum albumin (after a week of management) of male and female patients in success and failure in various form of parapneumonic effusion

Serumprotein				
Outcome	**Sex**	**Mean**	**N**	**Std. Deviation**
Failure	Male	2.2364	66	.35110
	Female	2.4385	13	.45559
	Total	2.2696	79	.37463
Success	Male	3.4025	40	.46271
	Female	3.5375	16	.38794
	Total	3.4411	56	.44345
Total	Male	2.6764	106	.69166
	Female	3.0448	29	.69209
	Total	2.7556	135	.70570

Albuminafterweek * outcome * sex Crosstabulation					
Count					
Sex			**Outcome**		**Total**
			Failure	**Success**	
Male	Albuminafterweek	albumin<3	66	3	69
		albumin>3	0	37	37
	Total		66	40	106
Female	Albuminafterweek	albumin<3	13	0	13
		albumin>3	0	16	16
	Total		13	16	29

Tab-5 Death and outcome

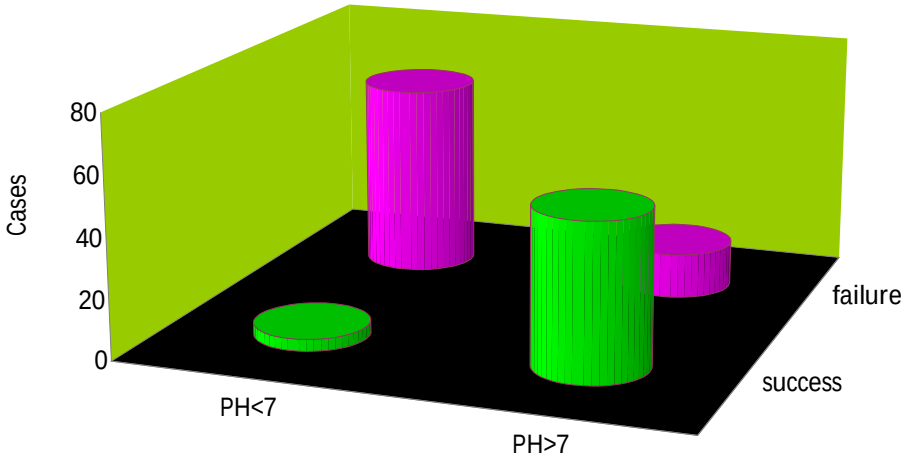
Death * outcome Crosstabulation				
Count				
		Outcome		**Total**
		Failure	**Success**	
	Alive	39	55	94
	Death	40	1	41
Total		**79**	**56**	**135**

Table -6 fever in success and failure and their test of significance

Fever			
	Observed N	**Expected N**	**Residual**
No fever	50	67.5	-17.5
Present	85	67.5	17.5
Total	**135**		

Outcome			
	Observed N	**Expected N**	**Residual**
Failure	79	67.5	11.5
Success	56	67.5	-11.5
Total	135		

Test Statistics		
	Fever	Outcome
Chi-Square	9.074[a]	3.919[a]
Df	1	1
Asymp. Sig.	.003	.048
a. 0 cells (.0%) have expected frequencies less than 5. The minimum expected cell frequency is 67.5.		



	PH<7	PH>7
success	4	52
failure	64	15

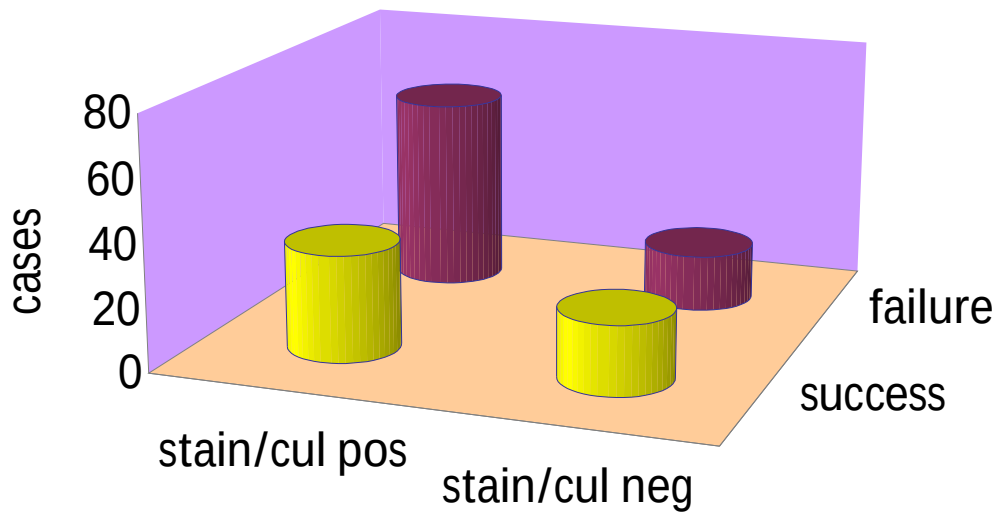
Graph depicting cross tabulation of success and failure with respesct to P_H

Results: Tab-7 univariate logistic regression analysis of P_H

Variables in the Equation							
		B(odd's)	**S.E.**	**Wald**	**Df**	**Sig.**	**Exp(B)**
Step 1[a]	PH(1)	-4.016	.593	45.876	1	.000	.018
	Constant	1.243	.293	17.993	1	.000	3.467
a. Variable(s) entered on step 1: PH.							

Table of univariate analysis depicting P_H with P value of 0.00 and odd 's of failure with low pH as 4.016.

Categorical Variables Codings			
		Frequency	**Parameter coding**
			(1)
Ph after 1 week of management	ph<7	68	1.000
	ph>7	67	.000



	stain/cul pos	stain/cul neg
success	34	22
failure	61	18

Graph depicting cross tabulation of success and failure with respesct to stain and culture positivity

Table (8) of univariate analysis depicting culture and staining positivity with P value of 0.40 and odd 's of failure with low pH as 0.785.

Classification Table[a,b]					
	Observed		**Predicted**		
			Outcome		**Percentage Correct**
			Failure	**Success**	
Step 0	outcome	Failure	79	0	100.0
		Success	56	0	.0
	Overall Percentage				58.5
a. Constant is included in the model.					
b. The cut value is .500					

Variables in the Equation							
		B	S.E.	Wald	Df	Sig.	Exp(B)
Step 1[a]	cultureposstain (1)	.785	.383	4.199	1	.040	2.193
	Constant	-.585	.214	7.459	1	.006	.557
a. Variable(s) entered on step 1: cultureposstain.							

Table(9) of univariate analysis depicting pleural fluid WBC count with P value of 0.00 and odd 's of failure with low pH as 4.277.

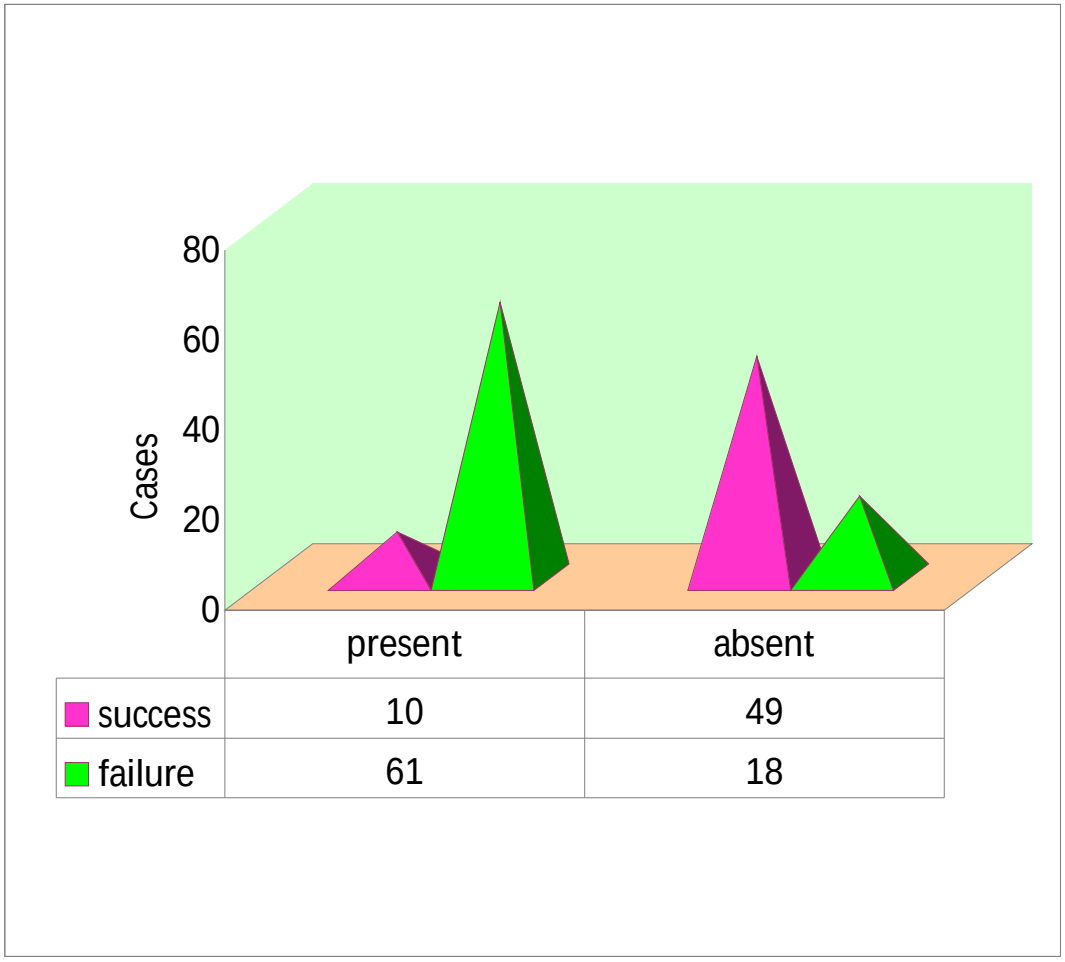
Categorical Variables Codings			
		Frequency	Parameter coding
			(1)
leucocount after 1 week	count<8000	56	1.000
	count>10000	79	.000

Variables in the Equation							
		B	**S.E.**	**Wald**	**Df**	**Sig.**	**Exp(B)**
Step 1[a]	Leukocyte(1)	4.277	.566	57.154	1	.000	72.000
	Constant	-2.331	.396	34.657	1	.000	.097
a. Variable(s) entered on step 1: leukocyte.							

Table (10) of univariate analysis depicting serum protein with P value of 0.00 and odd 's of failure with low protein as 7.412

Variables in the Equation							
		B	**S.E.**	**Wald**	**Df**	**Sig.**	**Exp(B)**
Step 1[a]	serumprotein	7.412	1.525	23.640	1	.000	1656.473
	Constant	-21.310	4.356	23.929	1	.000	.000
a. Variable(s) entered on step 1: serumprotein.							

Classification Table[a,b]					
	Observed		**Predicted**		
			Outcome		**Percentage Correct**
			Failure	**Success**	
Step 0	outcome	Failure	79	0	100.0
		Success	56	0	.0
	Overall Percentage				58.5
a. Constant is included in the model.					
b. The cut value is .500					



Graph depicting cross tabulation of success and failure with respesct to Presence and absence of loculation

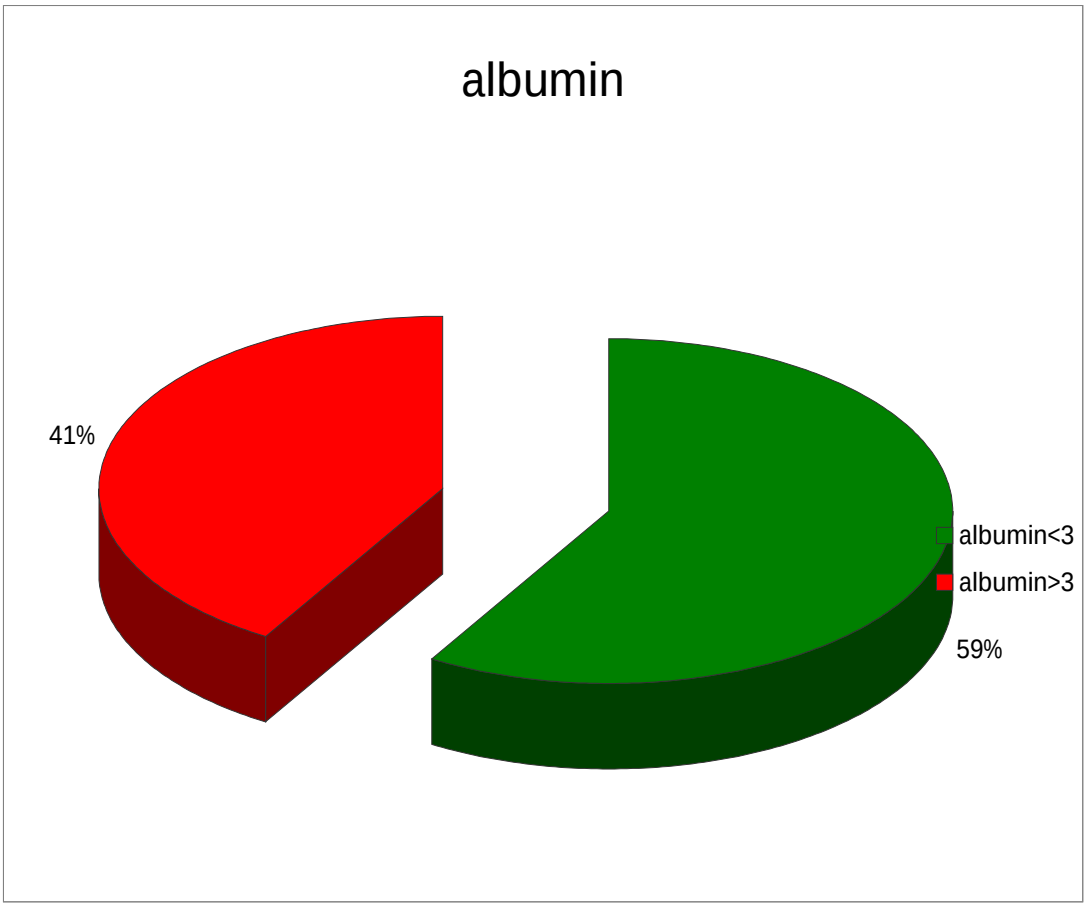
Table (11) of univariate analysis depicting loculation with P value of 0.00 and odd 's of failure with low pH as 3.166.

Variables in the Equation							
		B	**S.E.**	**Wald**	**Df**	**Sig.**	**Exp(B)**
Step 1[a]	loculation(1)	3.166	.485	42.626	1	.000	23.722
	Constant	-2.165	.399	29.432	1	.000	.115
a. Variable(s) entered on step 1: loculation.							

Table (12) of univariate analysis depictingfever with P value of 0.09 and odd ‘s of failure with persistant fever as 4.016.

Variables in the Equation							
		B	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**
Step 1[a]	Fever(1)	.952	.367	6.742	1	.009	2.591
	Constant	-.711	.231	9.488	1	.002	.491
a. Variable(s) entered on step 1: fever.							

Categorical Variables Codings			
		Frequency	**Parameter coding**
			(1)
Fever	No fever	50	1.000
	present	85	.000



Graph depicting the percentage of patients with albumin<3 and albumin>3 grams after treatment of complicated, complex parapneumonic and simple parapneumonic effusion

Table (13) Depicting different variables and their univariate logistic regression analysis.

Univariate logistic regression analysis

Variable	P value	Odd's	S.E
P_H	0.00	4.016	.593
WBC	0.00	4.277	.566
Loculation	0.00	3.166	.485
Fever	0.09	.711	.367
Serum proteins	0.00	7.412	1.525
Sex	0.095	0.708	0.424

Results show significant association with Ph, WBC, loculation, fever, serum proteins. Their odd were

Ph-4.0 loculation-3.1 fever-0.7

WBC-4.2 serum albumin-7.4

Table 14 multiple logistic regression analysis with analysis five independent variables. Method = backward stepwise (conditional)

		B	S.E.	Wald	Df	Sig.	Exp(B)
Step 1[a]	PH(1)	-5.610	2.573	4.753	1	.029	.004
	leukocyte(1)	1.756	1.376	1.627	1	.202	5.787
	loculation(1)	2.964	1.701	3.037	1	.081	19.377
	cultureposstain(1)	1.038	1.612	.414	1	.520	2.824
	Serumprotein	7.846	2.760	8.082	1	.004	2555.256
	Constant	-23.810	8.008	8.840	1	.003	.000
Step 2[a]	PH(1)	-5.599	2.459	5.183	1	.023	.004
	leukocyte(1)	1.674	1.401	1.427	1	.232	5.331
	loculation(1)	3.330	1.712	3.785	1	.052	27.931
	Serumprotein	7.907	2.835	7.780	1	.005	2715.493
	Constant	-23.840	8.198	8.457	1	.004	.000
Step 3[a]	PH(1)	-6.137	2.590	5.615	1	.018	.002
	loculation(1)	4.156	1.705	5.940	1	.015	63.785
	Serumprotein	8.788	3.112	7.974	1	.005	6555.751
	Constant	-25.585	8.953	8.166	1	.004	.000
a. Variable(s) entered on step 1: PH, leukocyte, loculation, cultureposstain, serumprotein.							

Table 15 : Logistic Regression

Block 1: Method = Forward Stepwise (Likelihood Ratio)

Variables in the Equation							
		B	**S.E.**	**Wald**	**Df**	**Sig.**	**Exp(B)**
Step 1[a]	PH(1)	-5.610	2.573	4.753	1	.029	.004
	leukocyte(1)	1.756	1.376	1.627	1	.202	5.787
	loculation(1)	2.964	1.701	3.037	1	.081	19.377
	cultureposstain(1)	1.038	1.612	.414	1	.520	2.824
	Serumprotein	7.846	2.760	8.082	1	.004	2555.256
	Constant	-23.810	8.008	8.840	1	.003	.000
Step 2[a]	PH(1)	-5.599	2.459	5.183	1	.023	.004
	leukocyte(1)	1.674	1.401	1.427	1	.232	5.331
	loculation(1)	3.330	1.712	3.785	1	.052	27.931
	Serumprotein	7.907	2.835	7.780	1	.005	2715.493
	Constant	-23.840	8.198	8.457	1	.004	.000
Step 3[a]	PH(1)	-6.137	2.590	5.615	1	.018	.002
	loculation(1)	4.156	1.705	5.940	1	.015	63.785
	Serumprotein	8.788	3.112	7.974	1	.005	6555.751
	Constant	-25.585	8.953	8.166	1	.004	.000
a. Variable(s) entered on step 1: PH, leukocyte, loculation, cultureposstain, serumprotein.							

INTERPRETATIONS

One hundred and thirty five patients with parapneumonic effusion were analysed during the study period .

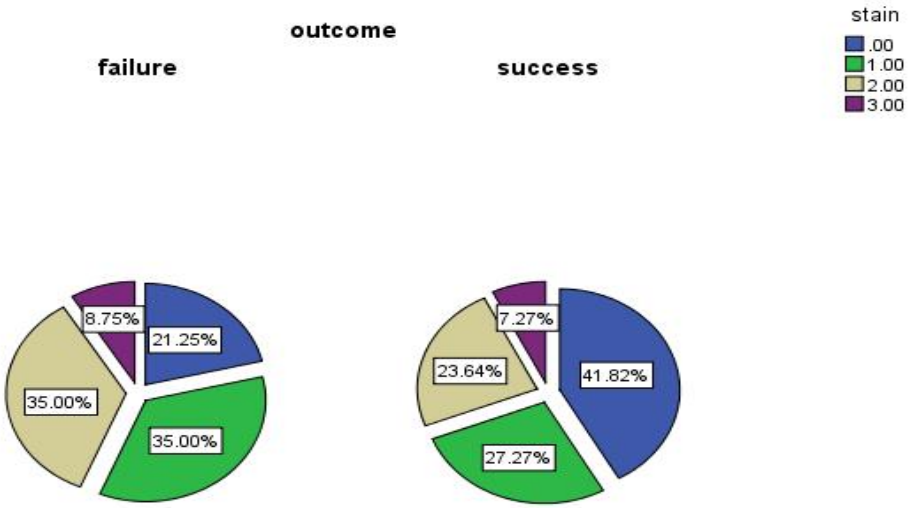
Ten with parapneumonic effusion had successful recovery treated with antibiotics according to culture and sensitivity pattern.

One hundred and thirty five patients with complicated and complex parapneumonic under tube thoracostomy with successful outcome in fifty six patients. Seventy nine patients with CPE had failed outcome.

Mortality was forty one (all cause) and CPE mortality was forty. Mortality percentage was 30.37%.

Various causes of parapneumonic effusion in the study were pneumonia-88, lung abscess-10, bronchietasis-16, unidentified causes(probability of pneumonia)-17.

Graph Staining Characteristics of Patients



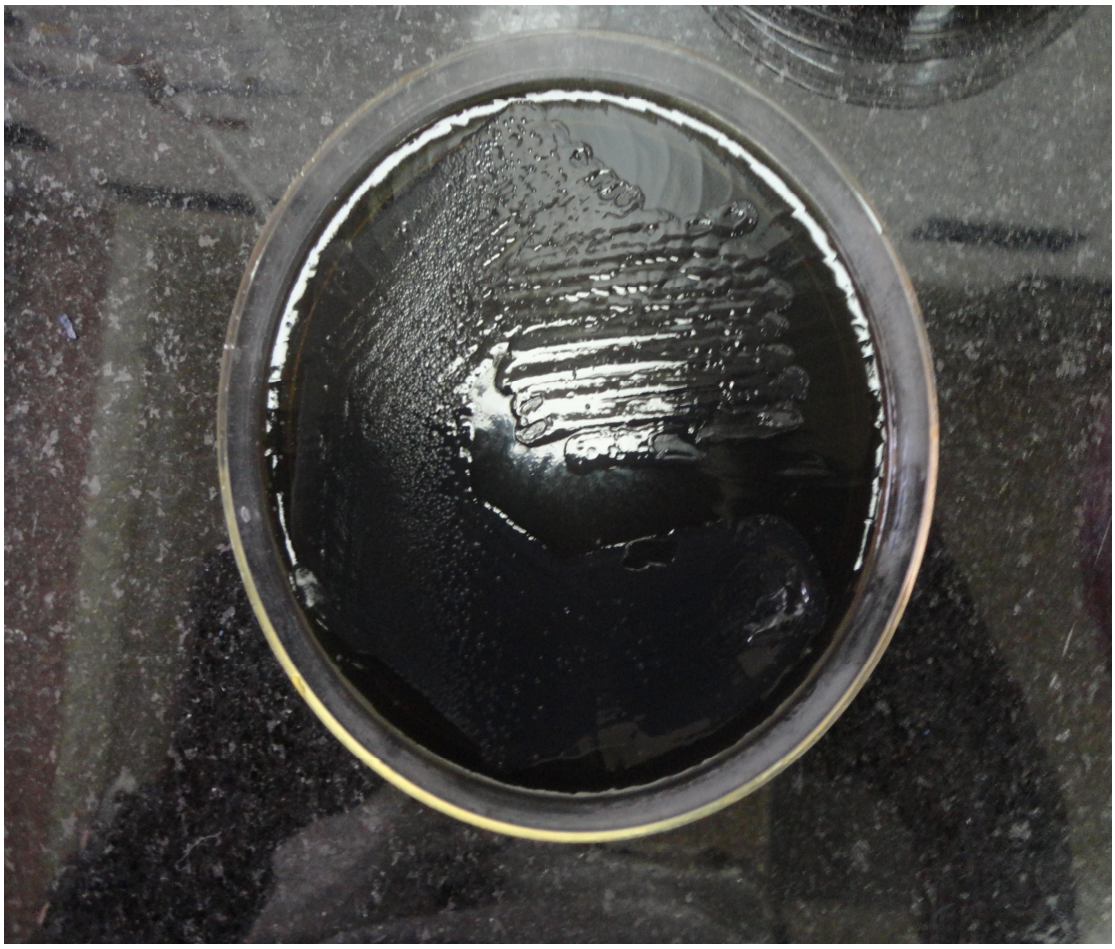
0.00= no growth

1.00= gram negative

2.00= gram positive

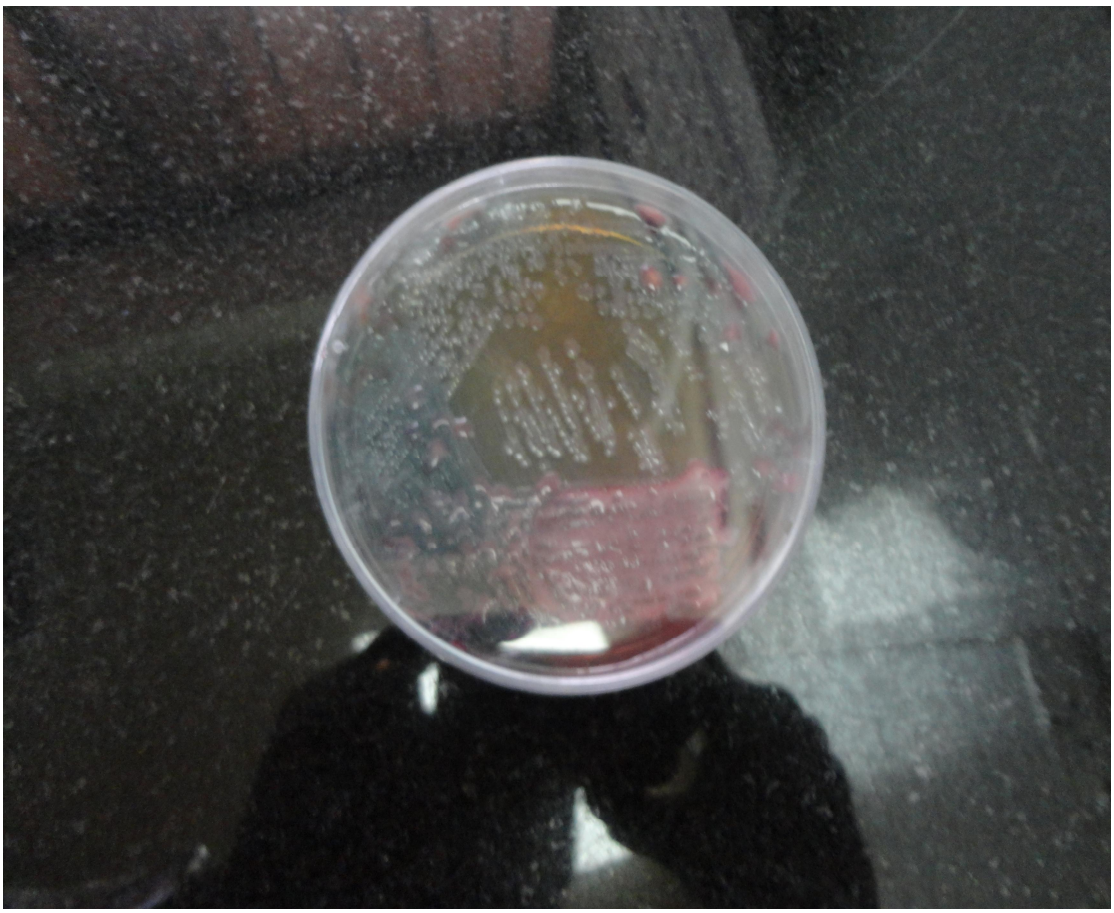
3.00= mixed infection.

PSEUDOMONAS GROWN IN CULTURE



PHOTOGRAPH SHOWING STAPHYLOCOCCUS AUREUS GROWN IN CULTURE IN BLOOD AGAR.

MIXED INFECTION WITH KLEBSIELLA AND PROTEUS SPECIES GROWN



Bacteriology of pleural space infection

Species	Stain	N
Nogrowth	.00	43
	Total	43
Klebsiella	1.00	11
	Total	11
Streptococi	2.00	28
	Total	28
Staphylococci	2.00	13
	Total	13
Pseudomonas	1.00	10
	Total	10
Mixed inf	3.00	11
	Total	11
Ecoli	1.00	7
	Total	7
Providentia	1.00	8
	Total	8
Acinetobacter	1.00	4
	Total	4
Total	.00	43
	1.00	40
	2.00	41
	3.00	11
	Total	135

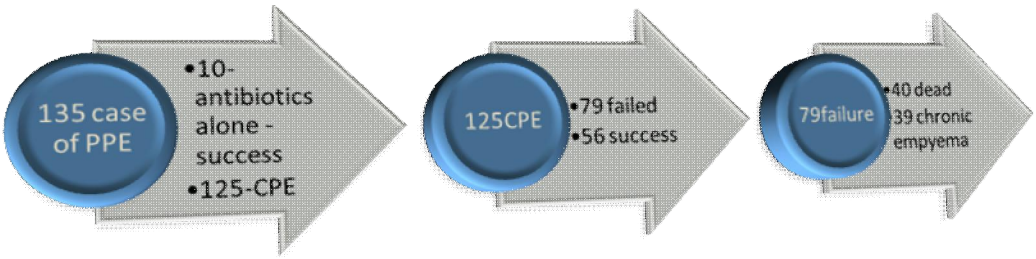
0.00= no growth 2.00= gram positive

1.00= gram negative. 3.00= mixed.

Causes of parapneumonic effusion	No of cases(N)
Pneumonia	88
Lung abscess	10
Bronchiectasis	16
Unidentified cause (high probabilityof pneumonia)	17

Of the 92 cases with stain &culture positivity in CPE - gram negative infection occurred in forty cases, forty one cases of gram positive infection occurred, eleven cases of mixed infection occurred, forty three cases had no growth but twenty eight cases had positivity with staining method probably denoting anaerobic infection.

Outcome of pleural space infection



Patients characteristics	Success (n=56)	Failure (n=79)	P value
Age	47.18±9.14	46.97±8.71	0.752
Sex (m/f)	40/16	66/13	0.00
Pleural fluid P_H	7.56±0.88	5.75±1.31	0.00
Wbc count	6406.27±2284.96	11887.24±3145	0.00
Protein	3.44±0.78	3.44±0.62	0.97
Serum albumin after week of treatment	3.44±0.44	2.26±0.37	0.01

Patient characteristics with P-value (tested by t-test).

Comparison of outcome in cases of complicated &complex parapneumonic effusion

- Mean age in success and failure were 47.18 ±9.14 & 46.97±8.4 and P value 0.752.
- Gender variation in success and failure was significant with P value of 0.00.
- In univariate analysis pleural fluid protein did not show significant results with mean protein in success and failure to be 3.44±0.62 and 3.44± 0.78 respectively with P value of 0.97.
- Mean H^+ ion in success was 7.56±0.88 while in failure was 5.75±1.31 p value of 0.00.

- Persistant fever after one week of antibiotics also had significant P value of 0.048
- Serum protein especially albumin in success and failure were 3.44±0.78 and 2.26±0.37 respectively with P value of o.oo.
- Other parameters such as co-morbid illness, fibrinpeel (paucity of cases) ,blood glucose did not show any significant results.

.

DISCUSSION

The success rate for conventional tube thoracostomy drainage is 32 to 71%.[86]

Success rate reported from other studies is comparable to that of 41% in our study.[88–90] High mortality rates from empyema have been reported, ranging from 1 to 61%.[87,91–93] In the present study, the overall mortality rate was 31% and 40 fatalities (30%) were directly related to empyemas. The duration of the pleural infection, the characteristics of the pleural fluid, the presence or absence of loculations the overall condition of the patient are the four critical important factors to be considered in the selection of a pleural drainage method. These four factors also influence the tube thoracostomy drainage outcome according to the review of Moran[95].

LeMense et al, [96] no difference in procedure success rates or hospital stay was observed between multiloculated and uniloculated empyemas, parapneumonic and nonparapneumonic empyemas, and culture proved and biochemically proved empyemas. Their success rate

of tube thoracostomy drainage was only 11%, because all patients had loculated pleural fluid at presentation.

In contrast, in one series of 26 patients who underwent thoracoscopy for chronic empyema of at least 3 weeks duration, over 50 percent had no evidence of intrapleural scar tissue – being still at the fibrinopurulent stage of their infection.[97] Success rate of tube thoracostomy without loculation in our study was around 86.7%.

Patients should be considered for surgery if they have ongoing signs of sepsis in association with a persistent pleural collection despite drainage and antibiotics. Failure of sepsis to resolve within 5-7 days[98] is suggested as an appropriate period following which a surgical opinion should be sought. Discussion with a thoracic surgeon should be considered in all cases failing to respond.

Prognosis in pleural infection

The long-term survival of patients with pleural infection is good if prompt treatment is initiated. In a series of 85 patients followed for up to 4 years, the mortality was 14% and all deaths occurred within the first 400 days after drainage[99]. Death was due to comorbid condition and not directly due to sepsis from the empyema. No reliable clinical, radiological

or pleural fluid characteristics accurately determine patients prognosis at initial presentation. Hypoalbuminaemia, the presence of loculated fluid and anaerobic infections have been related to adverse outcome in previous studies[100-101] although not in recent reports. Long-term sequelae of pleural empyema may include residual pleural thickening (up to 13% of patients).30 This is not usually associated with functional impairment although, rarely, extensive incapacitating pleural fibrosis may develop (fibrothorax).[102 103 104] Surgical decortication may occasionally provide symptomatic benefit for patients with a fibrothorax. Pleural calcification, bronchopleural fistula formation and development of empyema necessitans (disruption of the parietal pleura with spontaneous discharge of pleural contents evident under the chest wall) are other rare complications.

Early thoracotomy has the additional advantage that if decortications is accomplished within 2 weeks of pleural infection, the visceral pleural rind usually is easily extricated from the lung.[30]

Hence early referral of failing cases will benefit from surgery. The determinants such as PH (falling even after a week of management), loculation, serum albumin falling after a week of management can

successfully predict the failing cases according to our study which have proved statistically by using logistic regression model.

Limitation of study

Lack of anaerobic culture is the limitation of the study.

Summary of Results and Discussion

Table 7 to 12 shows univariate logistic regression analysis.

- Tab-7= Univariate analysis depicting P_H with P value of 0.00 and odd ‘s of failure with low pH as 4.016.
- Tab-8 = univariate analysis depicting culture and staining positivity with P value of 0.40 and odd ‘s of failure with low pH as 0.785.
- Table 9= univariate analysis depicting pleural fluid WBC count with P value of 0.00 and odd ‘s of failure with low pH as 4.277.
- Table 10 = univariate analysis depicting serum protein with P value of 0.00 and odd ‘s of failure with low protein as 7.412.
- Table (11) of univariate analysis depicting loculation with P value of 0.00 and odd ‘s of failure with low pH as 3.166.
- Table (12) of univariate analysis depicting fever with P value of 0.09 and odd ‘s of failure with persistant fever as 4.016.

Variables such as PH, loculation, positive culture, WBC count, serum albumin, fever had significant results in univariate analysis.

Table 14 & 15

- ❖ Multivariate logistic regression analysis showed significant results for P_H , loculation, serum protein in both forward Likelihood ratio and backward condition methods.
- ➢ P_H - 0.018 p value with odd's ratio of 6.14 , loculation – p value of 0.015 with odd's ratio of 4.15, serum albumin after weeks of antibiotic –P value of 0.004 with odd 8.78. in both the methods.

But multivariate analysis showed results for PH, loculation and serum albumin.(recommendation).

Thus making them the most important predictor of outcome of tube thoracostomy for CPE. It is recommended based upon this study that pleural P_H, loculation, serum albumin, be taken as markers determining the prognosis of parapneumonic effusion with regards to outcome.

CONCLUSION

To conclude determinants such P_H, pleural loculation, serum albumin can predict the outcome of pleural space infection especially in complicated & complex parapneumonic effusion. Such predictors can help to reduce the morbidity and mortality associated with complicated parapneumonic effusion by identification of failing cases and early referral for definite management.

The bacteriology of pleural space infection in our study is comparable to the bacteriology of similar studies with gram positive and gram negative organisms occurring equally in seventy percentage of infection.

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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-3

Title of the Work : The Bacteriology of Pleural Infection by standard methods and its effect on outcome in Tertiary Respiratory Care Center in India.

Principal Investigator : Dr.V.P.Arivudainambi
Designation : PG in MD (Pulmonary Medicine)
Department : Thoracic Medcine

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 07.05.2010 at the Modernised Seminar Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate form the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

SECRETARY
IEC, SMC, CHENNAI

CHAIRMAN,
IEC, SMC, CHENNAI

PROFORMA

Name:

Age: Sex: C.D. No:

Symptoms: Duration

Cough

Sputum

Breathlessness

Fever

Chest pain:

Past history:

- H/O
- Diabetes.
- Alcoholic liver disease
- Chronic lung diseases
- Hypertension
- IHD

Previous admission for effusion/empyema

INVESTIGATIONS

1.Pleural fluid biochemistry

a. protein

b. sugar

c.P_H

cytology – WBC count.

2.Complete blood count:

3. Blood glucose:

4.Liver function test:

a.SGOT d. Serum protein (albumin/globulin).

b. SGPT

c SALP

5. Sputum culture and sensitivity.

6. Ultrasonogram of chest.

7. CT SCAN

8. Thoracoscopy.

Antibiotics

KEY WORDS TO MASTER CHART

Hion- P_H in continous variable.

Stain cul pos- Both positive for staining and culture growth.

Fibrin peel- appearance of fibrin peel in thoracoscopy.

BC- total pleural fluid leucocyte count.

Serum protein – Albumin in continuous variable.

No.	Age	Sex	PG	Hion	PH	Protein	LEU	WBC	MR	Cul+	Locu	Fibrin	Fever	Pus	RBS	DM	OC	Alb	SP
1.	48	2	60.0	7.2	1.00	3.20	0	7,250	0	1	0	0	1	0	90.00	1	1	1.00	4.00
2.	49	1	50.0	5.8	0.00	3.10	1	11,920	0	1	1	1	1	1	146.00	2	0	0.00	2.20
3.	52	1	55.0	6.8	0.00	3.90	1	10,480	1	1	1	1	1	0	75.00	1	0	0.00	2.00
4.	39	1	90.0	8.2	1.00	3.70	0	7,500	0	1	0	0	1	0	100.00	1	1	1.00	3.30
5.	44	2	75.0	4.8	0.00	4.30	1	12,160	0	1	1	0	1	0	112.00	1	0	0.00	2.70
6.	63	1	60.0	7.6	1.00	3.00	0	7,000	0	1	0	0	1	0	176.00	2	0	0.00	2.40
7.	51	2	59.0	5.2	0.00	3.60	0	7,250	0	0	0	0	0	0	102.00	1	1	1.00	3.30
8.	29	1	85.0	8.9	1.00	3.60	0	6,750	0	0	0	0	0	0	106.00	1	1	1.00	3.50
9.	49	1	45.0	7.2	1.00	2.90	1	10,240	1	1	1	1	1	0	69.00	1	0	0.00	2.20
10.	35	1	40.0	6.0	0.00	2.50	1	12,400	1	0	0	0	1	0	79.00	1	0	0.00	2.30
11.	31	1	40.0	4.0	0.00	2.80	1	13,540	0	1	1	0	0	0	82.00	1	0	0.00	2.00
12.	42	1	80.0	5.0	0.00	3.10	1	10,000	0	0	1	0	1	0	95.00	1	0	0.00	2.00
13.	47	2	38.0	5.8	0.00	4.10	1	10,600	1	0	0	0	1	1	55.00	1	0	0.00	1.90
14.	55	1	60.0	7.0	1.00	3.20	1	10,100	0	0	0	0	0	0	73.00	1	1	0.00	3.30
15.	57	1	82.0	6.0	0.00	3.40	0	5,600	0	1	0	0	0	0	90.00	1	1	1.00	4.00
16.	52	1	43.0	7.0	1.00	5.60	1	10,100	0	1	1	0	1	0	160.00	2	0	0.00	2.00
17.	29	1	73.0	6.0	0.00	3.20	1	10,450	0	0	0	0	1	0	75.00	1	1	1.00	4.00
18.	64	1	72.0	7.4	1.00	4.20	1	11,320	0	1	0	0	0	0	77.00	1	1	1.00	3.00
19.	30	1	66.0	5.0	0.00	3.80	1	13,200	1	0	1	0	1	0	69.00	1	0	0.00	1.60
20.	44	1	39.0	6.0	0.00	3.80	1	10,200	0	1	1	0	1	0	49.00	1	0	0.00	2.10
21.	49	1	48.0	7.6	1.00	3.80	1	13,200	0	1	0	0	1	0	55.00	1	1	1.00	3.00
22.	50	2	66.0	8.2	1.00	3.60	1	11,200	0	1	1	0	0	0	68.00	1	0	0.00	2.60

No.	Age	Sex	PG	Hion	PH	Protein	LEU	WBC	MR	Cul+	Locu	Fibrin	Fever	Pus	RBS	DM	OC	Alb	SP
23.	43	2	55.0	8.7	1.00	3.80	0	4,900	0	1	0	0	0	0	65.00	1	1	1.00	4.00
24.	56	1	50.0	8.5	1.00	3.50	1	14,000	1	1	1	0	0	0	62.00	1	0	0.00	1.60
25.	48	1	39.0	8.4	1.00	3.90	1	13,800	0	1	1	0	0	0	59.00	1	0	0.00	2.60
26.	47	1	57.0	8.2	1.00	4.00	0	7,120	0	0	0	0	0	0	70.00	1	1	0.00	2.50
27.	43	1	55.0	5.4	0.00	2.60	1	12,800	1	1	0	0	1	0	56.00	1	0	0.00	2.30
28.	56	2	56.0	7.7	1.00	3.44	0	5,600	0	0	0	0	1	0	67.00	1	1	1.00	3.00
29.	52	1	65.0	9.1	1.00	3.90	1	11,200	0	1	1	0	1	0	171.00	2	0	0.00	2.20
30.	42	1	36.0	7.9	1.00	2.80	1	10,300	0	0	0	0	0	1	140.00	2	0	0.00	2.40
31.	47	2	48.0	7.2	1.00	3.10	0	6,700	0	1	0	0	1	0	52.00	1	1	1.00	3.70
32.	39	1	69.0	6.0	0.00	4.00	1	13,000	0	1	1	0	0	0	70.00	1	0	0.00	2.00
33.	41	1	46.0	7.0	1.00	3.10	0	5,900	0	0	1	0	0	0	62.00	1	1	1.00	3.60
34.	62	1	34.0	6.0	0.00	4.00	1	11,000	0	0	1	1	1	0	48.00	1	0	0.00	2.10
35.	53	1	80.0	5.8	0.00	6.40	1	17,800	1	1	1	1	1	1	180.00	2	0	0.00	1.50
36.	37	1	58.0	8.0	1.00	7.90	0	4,300	0	0	0	0	1	0	90.00	1	1	1.00	3.90
37.	46	1	56.0	5.6	0.00	3.40	1	15,300	0	1	1	0	1	0	61.00	1	0	0.00	2.00
38.	28	1	54.0	7.0	1.00	2.90	0	4,900	0	0	0	0	0	0	60.00	1	1	1.00	4.00
39.	29	1	45.0	6.6	0.00	3.10	1	10,800	1	1	1	0	1	1	50.00	1	0	0.00	2.10
40.	61	2	67.0	5.3	0.00	3.20	1	13,100	0	0	0	0	1	0	69.00	1	0	0.00	2.20
41.	51	1	51.0	4.1	0.00	3.50	1	11,500	0	0	1	1	1	0	55.00	1	0	0.00	2.90
42.	39	1	49.0	7.3	1.00	3.70	0	3,470	0	1	0	0	1	0	80.00	1	1	1.00	3.00
43.	41	1	51.0	4.7	0.00	4.10	1	17,900	0	1	1	1	1	1	80.00	1	0	0.00	2.70
44.	37	2	55.0	7.1	1.00	2.90	0	3,248	1	0	1	0	1	0	78.00	1	0	0.00	2.10

No.	Age	Sex	PG	Hion	PH	Protein	LEU	WBC	MR	Cul+	Locu	Fibrin	Fever	Pus	RBS	DM	OC	Alb	SP
45.	56	1	57.0	9.0	1.00	4.10	0	4,321	0	0	0	0	0	0	59.00	1	1	1.00	3.60
46.	50	1	32.0	3.8	0.00	4.10	1	10,700	0	1	1	0	0	0	50.00	1	0	0.00	2.60
47.	56	1	38.0	4.0	0.00	3.10	1	12,450	1	1	1	1	1	1	149.00	2	0	0.00	2.30
48.	42	2	42.0	7.0	1.00	3.40	0	2,489	0	1	0	0	1	0	61.00	1	1	1.00	3.60
49.	48	2	51.0	6.0	0.00	3.60	1	10,060	0	1	1	0	0	0	53.00	1	0	0.00	2.70
50.	49	1	51.0	3.7	0.00	3.80	1	12,600	1	0	0	0	1	0	51.00	1	0	0.00	2.20
51.	27	1	44.0	7.0	1.00	3.70	1	12,500	0	1	1	0	1	0	61.00	1	1	1.00	3.00
52.	28	1	45.0	6.3	0.00	2.70	1	10,210	1	1	0	0	0	0	151.00	2	0	0.00	2.60
53.	51	2	56.0	7.0	1.00	3.00	0	6,570	0	0	0	3	1	0	158.00	2	1	1.00	3.00
54.	39	2	72.0	8.4	1.00	3.90	0	5,700	0	1	0	3	1	0	167.00	2	1	1.00	4.00
55.	41	1	54.0	8.9	1.00	3.00	0	4,900	0	0	1	0	0	0	70.00	1	1	1.00	4.10
56.	49	1	72.0	5.6	0.00	4.30	1	17,400	0	1	1	1	1	0	74.00	1	0	0.00	2.10
57.	39	2	70.0	7.0	1.00	3.10	0	3,400	0	1	1	0	0	0	88.00	1	0	0.00	2.80
58.	55	1	56.0	6.4	0.00	4.00	1	10,100	1	0	1	0	1	0	71.00	1	0	0.00	1.50
59.	33	1	38.0	5.6	0.00	3.60	1	14,300	0	1	1	0	1	0	59.00	1	0	0.00	2.00
60.	33	1	39.0	7.0	1.00	3.20	0	8,000	0	0	1	0	0	0	57.00	1	1	1.00	4.00
61.	52	1	40.0	6.0	0.00	3.00	0	6,000	0	1	0	0	0	0	71.00	1	1	1.00	3.50
62.	56	1	44.0	7.0	1.00	3.00	1	10,200	1	0	0	0	1	0	57.00	1	0	0.00	2.20
63.	67	2	54.0	9.1	1.00	3.20	0	4,500	1	1	0	0	0	0	60.00	1	1	1.00	3.80
64.	48	1	49.0	4.8	0.00	3.50	1	12,000	0	0	0	0	0	0	49.00	1	0	0.00	2.10
65.	61	1	52.0	4.4	0.00	3.20	1	12,400	1	1	1	0	0	0	53.00	1	0	0.00	2.00
66.	50	1	39.0	3.3	0.00	2.90	1	12,000	1	0	0	0	1	0	50.00	1	0	0.00	1.90

No.	Age	Sex	PG	Hion	PH	Protein	LEU	WBC	MR	Cul+	Locu	Fibrin	Fever	Pus	RBS	DM	OC	Alb	SP
67.	46	1	49.0	3.2	0.00	3.40	1	10,900	0	1	1	0	0	0	50.00	1	0	0.00	2.50
68.	40	1	50.0	5.8	0.00	4.00	0	6,900	0	1	0	0	1	0	54.00	1	0	0.00	2.80
69.	46	2	43.0	8.0	1.00	4.20	0	5,400	0	1	1	0	1	0	57.00	1	1	1.00	3.50
70.	70	1	36.0	6.2	0.00	3.10	1	13,800	0	1	1	0	1	0	41.00	1	0	0.00	2.70
71.	55	1	36.0	5.9	0.00	3.40	0	1,330	1	1	0	0	1	0	40.00	1	0	0.00	2.60
72.	46	1	36.0	5.8	0.00	3.20	1	13,000	0	1	1	0	0	0	46.00	1	0	0.00	2.50
73.	48	1	46.0	6.7	0.00	4.10	1	14,000	0	0	0	0	1	0	48.00	1	0	0.00	2.70
74.	45	1	48.0	7.0	1.00	4.20	0	7,000	0	1	0	0	0	0	49.00	1	1	1.00	3.00
75.	43	1	48.0	8.0	1.00	4.50	0	4,800	0	1	0	0	1	0	56.00	1	1	1.00	3.20
76.	46	1	43.0	8.0	1.00	4.20	0	5,400	1	1	0	0	0	0	49.00	1	0	0.00	2.50
77.	50	1	52.0	5.6	0.00	3.60	1	12,000	0	1	1	0	0	0	80.00	0	0	0.00	2.30
78.	51	1	50.0	5.6	0.00	4.10	1	13,000	0	1	0	0	1	0	79.00	1	0	0.00	2.10
79.	61	1	54.0	7.0	1.00	2.90	0	7,010	0	1	0	0	1	0	78.00	1	1	1.00	3.60
80.	37	1	67.0	6.2	0.00	2.90	1	10,410	1	0	1	0	0	0	102.00	1	0	0.00	2.10
81.	43	1	52.0	8.9	1.00	3.10	0	7,300	0	0	0	0	0	0	100.00	1	1	1.00	3.30
82.	39	2	61.0	9.8	1.00	3.10	0	6,200	0	0	1	0	0	0	101.00	1	1	1.00	3.00
83.	41	1	49.0	4.3	0.00	3.30	1	11,000	1	1	1	1	1	1	58.00	1	0	0.00	2.20
84.	62	1	52.0	7.4	1.00	3.80	0	5,300	0	0	0	0	1	0	140.00	2	1	1.00	3.00
85.	48	1	49.0	6.0	0.00	3.50	1	12,000	0	0	0	0	0	0	61.00	1	0	0.00	2.10
86.	38	1	51.0	7.0	1.00	3.20	1	11,200	0	1	0	0	0	0	60.00	1	1	1.00	3.40
87.	51	1	36.0	4.8	0.00	3.10	1	18,020	1	1	1	1	1	1	58.00	1	0	0.00	2.10
88.	57	1	56.0	8.1	1.00	4.00	0	8,000	0	1	0	0	0	0	57.00	1	1	1.00	3.30

No.	Age	Sex	PG	Hion	PH	Protein	LEU	WBC	MR	Cul+	Locu	Fibrin	Fever	Pus	RBS	DM	OC	Alb	SP
89.	40	1	41.0	5.6	0.00	3.20	1	15,400	1	1	1	0	0	0	55.00	1	0	0.00	1.50
90.	46	1	50.0	5.2	0.00	3.20	1	13,200	1	1	1	0	0	0	169.00	2	0	0.00	2.10
91.	28	1	55.0	4.0	0.00	3.00	1	11,800	1	1	1	1	1	0	102.00	1	0	0.00	2.00
92.	39	1	57.0	4.6	0.00	3.00	1	14,200	1	1	1	0	1	0	110.00	1	0	0.00	2.00
93.	41	1	54.0	4.8	0.00	3.00	1	12,600	1	1	1	0	1	0	102.00	1	0	0.00	1.90
94.	42	2	39.0	5.0	0.00	3.70	1	10,200	0	0	0	0	1	0	100.00	1	0	0.00	1.70
95.	50	1	51.0	3.9	0.00	4.00	1	10,300	1	1	1	1	1	1	109.00	1	0	0.00	1.90
96.	29	1	68.0	9.0	1.00	3.00	0	4,521	0	1	0	0	0	0	108.00	1	1	0.00	3.70
97.	55	1	59.0	7.0	1.00	3.80	0	3,600	0	0	0	0	1	0	100.00	1	1	1.00	3.30
98.	53	1	67.0	6.9	0.00	2.70	1	14,700	1	1	1	0	1	0	97.00	1	0	0.00	2.70
99.	53	1	56.0	7.0	1.00	3.00	0	5,400	0	0	0	0	1	0	91.00	1	1	1.00	3.80
100.	40	2	63.0	6.0	0.00	3.10	1	10,900	1	1	1	0	1	0	79.00	1	0	0.00	3.40
101.	57	1	56.0	7.0	1.00	3.00	1	11,200	0	1	0	0	1	0	96.00	1	1	1.00	3.50
102.	45	1	64.0	5.0	0.00	3.10	1	11,200	1	1	1	0	1	0	89.00	1	0	0.00	2.20
103.	51	1	36.0	6.7	0.00	3.10	1	18,040	0	1	1	0	1	0	79.00	1	0	0.00	2.60
104.	57	1	56.0	6.5	0.00	3.60	1	14,200	1	1	1	0	1	0	89.00	1	0	0.00	2.50
105.	49	1	63.0	7.0	1.00	3.80	0	4,520	0	1	0	0	1	0	88.00	1	1	1.00	3.00
106.	47	1	39.0	6.5	0.00	3.40	1	11,000	1	1	1	0	1	0	81.00	1	0	0.00	2.10
107.	50	2	60.0	6.4	0.00	2.90	1	10,900	0	1	1	0	1	0	89.00	1	0	0.00	2.60
108.	39	1	63.0	6.2	0.00	3.20	1	10,700	1	1	1	0	1	0	79.00	1	0	0.00	1.60
109.	61	1	63.0	7.0	1.00	3.00	0	6,400	0	1	0	0	1	0	104.00	1	1	1.00	2.00
110.	58	1	67.0	7.2	1.00	3.20	0	4,800	0	1	0	0	1	0	99.00	1	1	1.00	3.70

No.	Age	Sex	PG	Hion	PH	Protein	LEU	WBC	MR	Cul+	Locu	Fibrin	Fever	Pus	RBS	DM	OC	Alb	SP
111.	42	1	62.0	4.2	0.00	3.00	1	10,700	1	1	1	0	1	0	71.00	1	0	0.00	2.40
112.	47	1	53.0	7.8	1.00	3.00	0	6,300	0	0	0	0	1	0	120.00	1	1	1.00	3.00
113.	49	1	86.0	4.8	0.00	3.00	1	12,400	1	1	1	0	1	0	134.00	1	0	0.00	2.80
114.	54	1	58.0	7.0	1.00	3.70	0	4,000	0	0	0	0	1	0	108.00	1	1	1.00	3.00
115.	55	1	66.0	6.4	0.00	3.00	1	12,000	0	1	1	0	0	0	101.00	1	0	0.00	2.70
116.	49	1	77.0	7.2	1.00	3.00	1	13,900	1	1	1	0	1	0	100.00	1	0	0.00	2.70
117.	47	1	58.0	7.4	1.00	3.10	0	3,800	0	1	1	0	1	0	102.00	1	1	1.00	3.00
118.	54	1	59.0	7.5	1.00	3.00	0	5,300	0	0	0	0	0	0	98.00	1	1	1.00	3.10
119.	50	1	69.0	5.6	0.00	2.90	1	15,700	1	1	1	0	1	0	79.00	1	0	0.00	2.70
120.	51	1	55.0	7.0	1.00	3.60	1	15,900	1	1	1	0	1	0	61.00	1	0	0.00	2.80
121.	49	2	67.0	6.4	0.00	3.20	1	16,300	1	1	1	0	1	0	79.00	1	0	0.00	2.20
122.	56	2	43.0	5.1	0.00	3.00	1	12,349	0	1	1	0	1	0	56.00	1	0	0.00	2.70
123.	37	2	59.0	7.0	1.00	3.80	0	4,590	0	1	1	0	0	0	102.00	1	0	0.00	2.10
124.	65	1	67.0	3.1	0.00	2.90	1	14,780	0	1	1	0	1	0	105.00	1	0	0.00	2.00
125.	41	1	37.0	3.1	0.00	4.10	1	12,370	0	1	1	0	1	0	106.00	1	0	0.00	2.50
126.	50	1	70.0	7.6	1.00	3.00	0	4,890	0	1	0	0	0	0	101.00	1	1	1.00	4.00
127.	53	2	71.0	7.5	1.00	4.10	0	5,600	0	1	0	0	1	0	89.00	1	1	1.00	4.00
128.	45	2	71.0	7.8	1.00	3.80	0	6,600	0	1	0	0	0	0	102.00	1	1	1.00	3.80
129.	29	1	67.0	7.0	1.00	2.90	0	7,000	0	0	0	0	1	0	98.00	1	1	1.00	4.00
130.	39	2	69.0	8.0	1.00	2.70	0	5,480	0	1	0	0	0	0	88.00	1	1	1.00	3.60
131.	60	1	78.0	8.3	1.00	1.90	0	5,380	0	1	0	0	0	0	79.00	1	1	1.00	3.30
132.	51	2	85.0	8.1	1.00	3.00	0	5,250	0	1	0	0	1	0	89.00	1	1	1.00	3.20

No.	Age	Sex	PG	Hion	PH	Protein	LEU	WBC	MR	Cul+	Locu	Fibrin	Fever	Pus	RBS	DM	OC	Alb	SP
133.	59	1	69.0	9.0	1.00	2.80	0	6,210	0	1	0	0	0	0	86.00	1	1	1.00	4.00
134.	57	2	73.0	7.3	1.00	2.80	0	5,000	0	1	0	0	0	0	120.00	1	1	1.00	3.10
135.	51	1	75.0	7.1	1.00	2.90	0	8,000	0	1	0	0	0	0	80.00	1	1	1.00	3.60

Key

Sex

1 = Male

2 = Female

Mortality (MR)

0 = Alive

1 = Death

Culture Positive (Cul+)

1 = Growth

0 = No Growth

Loculation (Locu)

1 = Present

0 = Absent

PH

1 - > 7

0 - < 7

Diabetes (DM)

1 = Absent

2 = Present

Outcome (OC)

1 = Success

0 = Failure

Albumin (Alb)

0 - < 3

1 - > 3

Leucocyte (LEU)

1 - > 10000

0 - < 8000

Sl.No.	Positivestaincul	Outcome	Species	Stain
1.	staingrowth	success	klebsiella	1.00
2.	staingrowth	failure	pseudomonas	1.00
3.	staingrowth	failure	providentia	1.00
4.	staingrowth	success	klebsiella	1.00
5.	staingrowth	failure	pseudomonas	1.00
6.	staingrowth	failure	acinetobacter	1.00
7.	nogrowth	success	nogrowth	0.00
8.	nogrowth	success	nogrowth	0.00
9.	staingrowth	failure	streptococi	2.00
10.	nogrowth	failure	nogrowth	0.00
11.	staingrowth	failure	streptococi	2.00
12.	nogrowth	failure	nogrowth	0.00
13.	nogrowth	success	nogrowth	0.00
14.	staingrowth	success	streptococi	2.00
15.	nogrowth	success	nogrowth	0.00
16.	staingrowth	failure	streptococi	2.00
17.	nogrowth	success	nogrowth	0.00
18.	staingrowth	success	staphylococci	2.00
19.	nogrowth	failure	nogrowth	0.00
20.	staingrowth	failure	klebsiella	1.00
21.	staingrowth	success	klebsiella	1.00
22.	staingrowth	failure	staphylococci	2.00
23.	staingrowth	failure	streptococi	2.00
24.	nogrowth	failure	nogrowth	0.00
25.	staingrowth	failure	mixed inf	3.00
26.	nogrowth	success	nogrowth	0.00
27.	staingrowth	failure	ecoli	1.00
28.	nogrowth	success	nogrowth	0.00
29.	staingrowth	failure	providentia	1.00

Sl.No.	Positivestaincul	Outcome	Species	Stain
30.	nogrowth	failure	nogrowth	0.00
31.	staingrowth	success	ecoli	1.00
32.	staingrowth	failure	staphylococci	2.00
33.	nogrowth	success	nogrowth	0.00
34.	nogrowth	failure	nogrowth	0.00
35.	staingrowth	failure	streptococi	2.00
36.	nogrowth	success	nogrowth	0.00
37.	staingrowth	failure	streptococi	2.00
38.	nogrowth	success	nogrowth	0.00
39.	staingrowth	failure	staphylococci	2.00
40.	nogrowth	failure	streptococi	2.00
41.	nogrowth	failure	nogrowth	0.00
42.	staingrowth	success	mixed inf	3.00
43.	staingrowth	failure	providentia	1.00
44.	nogrowth	failure	nogrowth	0.00
45.	nogrowth	success	nogrowth	0.00
46.	staingrowth	failure	acinetobacter	1.00
47.	staingrowth	failure	ecoli	1.00
48.	staingrowth	success	klebsiella	1.00
49.	staingrowth	failure	pseudomonas	1.00
50.	nogrowth	failure	nogrowth	0.00
51.	staingrowth	success	mixed inf	3.00
52.	staingrowth	failure	streptococi	2.00
53.	nogrowth	success	nogrowth	0.00
54.	staingrowth	success	streptococi	2.00
55.	nogrowth	success	nogrowth	0.00
56.	staingrowth	failure	staphylococci	2.00
57.	staingrowth	failure	streptococi	2.00
58.	nogrowth	failure	nogrowth	0.00

Sl.No.	Positivestaincul	Outcome	Species	Stain
59.	staingrowth	failure	klebsiella	1.00
60.	nogrowth	success	nogrowth	0.00
61.	staingrowth	success	pseudomonas	1.00
62.	nogrowth	failure	nogrowth	0.00
63.	staingrowth	success	mixed inf	3.00
64.	nogrowth	failure	nogrowth	0.00
65.	staingrowth	failure	ecoli	1.00
66.	nogrowth	failure	nogrowth	0.00
67.	staingrowth	failure	providentia	1.00
68.	staingrowth	failure	acinetobacter	1.00
69.	staingrowth	success	providentia	1.00
70.	staingrowth	failure	ecoli	1.00
71.	staingrowth	failure	providentia	1.00
72.	staingrowth	failure	streptococi	2.00
73.	nogrowth	failure	nogrowth	0.00
74.	staingrowth	success	staphylococci	2.00
75.	staingrowth	success	klebsiella	1.00
76.	staingrowth	failure	streptococi	2.00
77.	staingrowth	failure	streptococi	2.00
78.	staingrowth	failure	staphylococci	2.00
79.	staingrowth	success	streptococi	2.00
80.	nogrowth	failure	nogrowth	0.00
81.	nogrowth	success	nogrowth	0.00
82.	nogrowth	success	nogrowth	0.00
83.	staingrowth	failure	pseudomonas	1.00
84.	nogrowth	success	nogrowth	0.00
85.	nogrowth	failure	nogrowth	0.00
86.	staingrowth	success	streptococi	2.00
87.	staingrowth	failure	klebsiella	1.00

Sl.No.	Positivestaincul	Outcome	Species	Stain
88.	staingrowth	success	staphylococci	2.00
89.	staingrowth	failure	streptococi	2.00
90.	staingrowth	failure	providentia	1.00
91.	nogrowth	failure	nogrowth	0.00
92.	staingrowth	failure	mixed inf	3.00
93.	staingrowth	failure	pseudomonas	1.00
94.	nogrowth	failure	nogrowth	0.00
95.	staingrowth	failure	mixed inf	3.00
96.	staingrowth	failure	mixed inf	3.00
97.	nogrowth	success	nogrowth	0.00
98.	staingrowth	failure	streptococi	2.00
99.	nogrowth	success	nogrowth	0.00
100.	staingrowth	failure	staphylococci	2.00
101.	staingrowth	success	streptococi	2.00
102.	staingrowth	failure	streptococi	2.00
103.	staingrowth	failure	klebsiella	1.00
104.	staingrowth	failure	pseudomonas	1.00
105.	nogrowth	success	nogrowth	0.00
106.	staingrowth	failure	streptococi	2.00
107.	staingrowth	failure	mixed inf	3.00
108.	staingrowth	failure	staphylococci	2.00
109.	staingrowth	success	providentia	1.00
110.	nogrowth	success	nogrowth	0.00
111.	staingrowth	failure	ecoli	1.00
112.	nogrowth	success	nogrowth	0.00
113.	staingrowth	failure	mixed inf	3.00
114.	nogrowth	success	nogrowth	0.00
115.	staingrowth	failure	pseudomonas	1.00
116.	staingrowth	failure	streptococi	2.00

Sl.No.	Positivestaincul	Outcome	Species	Stain
117.	staingrowth	success	staphylococci	2.00
118.	nogrowth	success	nogrowth	0.00
119.	staingrowth	failure	klebsiella	1.00
120.	staingrowth	failure	streptococi	2.00
121.	staingrowth	failure	mixed inf	3.00
122.	staingrowth	failure	streptococi	2.00
123.	staingrowth	failure	staphylococci	2.00
124.	staingrowth	failure	streptococi	2.00
125.	staingrowth	failure	pseudomonas	1.00
126.	staingrowth	success	streptococi	2.00
127.	staingrowth	success	staphylococci	2.00
128.	staingrowth	success	pseudomonas	1.00
129.	staingrowth	success	streptococi	2.00
130.	staingrowth	success	mixed inf	3.00
131.	staingrowth	success	pseudomonas	1.00
132.	staingrowth	success	streptococi	2.00
133.	staingrowth	success	klebsiella	1.00
134.	staingrowth	success	ecoli	1.00
135.	nogrowth	success	nogrowth	0.00

Key

0.0 = No growth

1.0 = gram positive

2.0 = gram negative

3.0 = Mixed Growth

CONSENT FORM

1) I AGREE TO PARTICIPATE IN STUDY TITLED" THE BACTERIOLOGY OF PLEURAL SPACE INFECTION AND CLINICAL, LABORATORY AND PHYSICAL DETERMINANTS OF OUTCOME OF INFECTION"

2) I CONFIRM THAT I HAVE BEEN TOLD ABOUT THIS STUDY IN MY MOTHER TONGUE & HAVE HAD THE OPPURTUNITY TO ASK QUESTIONS

3) I UNDERSTAND THAT MY PARTIPATION IS VOLUNTERY & I MAY REFUSE TO PARTICIPATE AT ANY TIME WITHOUT GIVING ANY REASON AND WITHOUT AFFECTING MY BENEFITS.

4) I AGREE NOT TO RESTRICT THE USE OF ANY DATE OR RESULTS THAT ARISE FROM THE STUDY

5) I AGREE TO UNDERGO THORACENTESIS TUBE THORACOSTOMY WHICH IS PART OF MANAGEMENT TOOL FOR MY DISEASE

NAME OF THE PARTICIPANT:

SIGNATURE/THUMB PRINT

INVESTIGATOR

ABSTRACT

Study objectives: To describe bacteriology of pleural space infection and determine the factors that would predict the outcome of the infection in various classes of parapneumonic effusion.

Study design: Prospective review of patients admitted to the tertiary respiratory care hospital from june 2009 to june 2011 with the diagnosis of simple, complicated and complex parapneumonic effusion.

Materials and methods : Patients features such as Age ,sex, pleural fluid protein, WBC, P_H , Glucose, pleural loculation, co-morbid illness like diabetes, Pleural fluid positivity for gram stain and culture growth, mortality, serum albumin levels after a week treatment, effusion quantity were analysed to identify the determinants of outcome in Complicated parapneumonic Effusion. To define the bacteriology of pleural space infection pleural fluid gram staining and growth in culture were analysed. The data were compared between two outcomes success and failure and statistically analysis using multiple logistic regression done .

Results: Of the one hundred thirty five diagnosis of simple parapneumonic ten had complete resolution of symptoms. Out of the reamaining ,one hundred twenty five cases of parapneumonic effusion . Seventy nine had failed outcome and fifty six had successful outcome.

univariate and multivariate logistic regression used. Bacteriology of pleural space infection showed both gram positive and gram negative growth comparable results to other studies.. Univariate analysis showed P_H, loculation , positive stain , culture, fever, serumalbumin to be statistically significant. Multivariate analysis showed PH, loculation, serumalbumin were important determinants predicting either success or failure of tube thoracostomy.

Conclusion; By multivariate analysis pleural fluid P_H, loculation , serum albumin were predictive factors for outcome of complicated and complex parapneumonic effusion. They could be used for reffering the cases for definitive management.

Key words: bacteriology , pleural infection, outcome .